

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 61

Editor

ARTHUR W. PROETZ, M.D.

Beaumont Building, St. Louis, 8

Associate Editor

BERNARD J. McMAHON, M.D.

Missouri Theatre Building, St. Louis, 3

1

Editorial Board

L. R. BOIES, M.D. . . . Minneapolis
LOUIS H. CLERF, M.D. . . . Philadelphia
SAMUEL J. CROWE, M.D. . . . Baltimore
EDMUND P. FOWLER, JR., M.D., New York
DAVID R. HIGBEE, M.D. . . . San Diego, Calif.
ANDERSON C. HILDING, M.D. . . . Duluth
FREDERICK T. HILL, M.D. . . . Waterville, Me.

HAROLD I. LILLIE, M.D. . . . Rochester, Minn.
JOHN G. McLAURIN, M.D. . . . Dallas
LEROY A. SCHALL, M.D. . . . Boston
BEN H. SENTURIA, M.D. . . . St. Louis
FRANCIS A. SOOY, M.D. . . . San Francisco
H. MARSHALL TAYLOR, M.D., Jacksonville, Fla.
O. E. VAN ALYEA, M.D. . . . Chicago

1

Published Quarterly

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE 1 1010 BEAUMONT BUILDING, 8

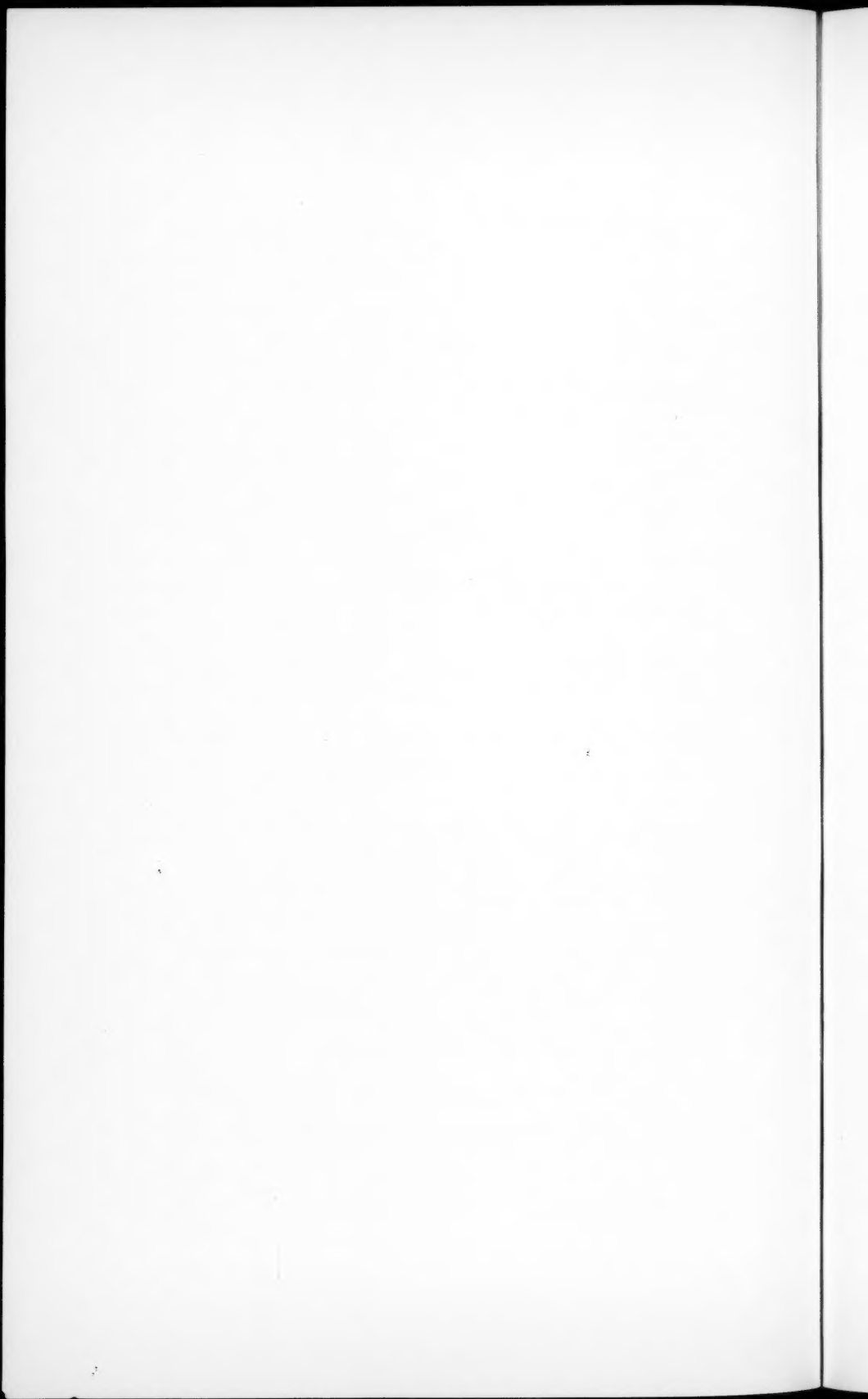
BUSINESS OFFICE 1 P. O. BOX 1345, CENTRAL STATION, 1

ST. LOUIS, MO., U.S.A.

COPYRIGHT, 1952

ANNALS PUBLISHING COMPANY

Annual Subscription in United States, Spain, Central and South America, \$10.00 in Advance.
Canada, \$10.20. Other Countries, \$10.80.



Contents.

	PAGE
LXXVI—Aerotitis Media. A Critical Review. Reed W. Hyde, USAF (MC), Randolph Field, Texas	937
LXXVII—Problems in Differential Diagnosis of Lesions of the Lower Portion of the Esophagus and the Cardia. Herman J. Moersch, M.D., Rochester, Minn.	976
LXXVIII—The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System. M. R. Dix, M.D., and C. S. Hallpike, M.D., London, England	987
LXXIX—Massive Osteoma of the Ethmoid Sinus. Marvin J. Tamari, M.D., and Edward B. Weisman, M.D., Chicago, Ill.	1017
LXXX—Some Principles in the Structure of Vibratile Cilia. H. Engström, M.D., and J. Wersäll, M.D., Stockholm, Sweden	1027
LXXXI—History of Tracheotomy. Robert E. Priest, M.D., Minneapolis, Minn.	1039
LXXXII—A Prolonged Local Anaesthetic in Control of Post-Tonsillectomy Pain. Morris Davidson, M.D., St. Louis, Mo., Robert G. Boles, M.D., Louisville, Ky., and Sanford C. Snyderman, M.D., Fort Wayne, Ind.	1046
LXXXIII—Cysts of the Thyroglossal Duct as a Complicating Factor in Laryngeal Operations. Frederick A. Figi, M.D., and Hugh A. Johnson, M.D., Rochester, Ind.	1048
LXXXIV—The Treatment of Deafness by Prothesis. H. G. Kobrak, M.D., Chicago, Ill.	1053
LXXXV—"Clocking" of the Natis as an Aid in Demonstrating Rhinoplasty. A. P. Seltzer, M.D., ScD., Philadelphia, Pa.	1067
LXXXVI—The Surgical Treatment of Laryngoceles. With Report of a Case. John J. O'Keefe, M.D., Philadelphia, Pa.	1071
LXXXVII—Surgery in Intra-Oral Cancer. Maurice F. Snitman, M.D., Chicago, Ill.	1076
LXXXVIII—Obstruction of the Air Passages. Wendell A. Weller, Colonel, MC, U. S. Army, San Francisco, Calif.	1080
LXXXIX—Treatment of Malignancy of the Buccal Mucous Membrane, Gingiva, Soft and Hard Palate. James W. Hendrick, M.D., San Antonio, Texas	1094
The Scientific Papers of the American Broncho-Esophagological Association	
XC—Traumatic Rupture of the Lower Trachea with Stenosis. A Case Report. G. Arnold Henry, M.D., Toronto, Canada	1114
XCI—Stenosis of the Esophagus in Benign Mucous Membrane Pemphigus. Edward B. Benedict, M.D., and Walter F. Lever, M.D. (By Invitation), Boston, Mass.	1120
XCII—Blind Bouginage in the Treatment of Benign Esophageal Obstruction. Lyman Richards, M.D., and Herbert J. Dietrich, Jr., M.D. (By Invitation), Brookline, Mass.	1134

CONTENTS—Continued

	PAGE
XCIII—Esophagitis: A Clinical Evaluation. Walter B. Hoover, M.D., Boston, Mass.	1148
XCIV—Congenital Malformations of the Trachea, Bronchi and Lung. Paul H. Holinger, M.D., Kenneth C. Johnston, M.D., Victor Parchet, M.D., and Arnold A. Zimmermann, Dr. es Sc., Chicago, Ill.	1159
XCV—Observations on Bronchial Movements and Elasticity by Means of a Recording Bronchial Caliper. Albert H. Andrews, Jr., M.D., Chicago, Ill.	1181
XCVI—Hidden or Unsuspected Bronchiectasis in the Asthmatic. Richard H. Overholt, M.D., and James H. Walker, M.D., Boston, Mass.	1198
XCVII—Fat in the Tracheo-Bronchial Tree with Report of a Case of True Lipoma of the Bronchus. Alexander H. Beaton, Capt. M. C., and Clyde A. Heatly, M.D., Rochester, N. Y.	1206
Society Proceedings	
Chicago Laryngological and Otolological Society, Meeting of Monday, March 3, 1952. The Otolaryngologist in a Hearing Conservation Program—Otogenic Intracranial Complications in the Antibiotic Era.	1216
Meeting of Monday, April 7, 1952. Thyrotomy for Arytenoidectomy in Bilateral Abductor Paralysis of the Vocal Cords—Nasal Implants in Children and in Adults, with Preliminary Note On the Use of Ox Cartilage	1220
Abstracts of Current Articles	1222
Books Received	1229
Notices	1230
Officers of the National Otolaryngological Societies	1232
Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association	1233
Index of Authors	1234
Index of Titles	1236

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

VOL 61

DECEMBER, 1952

No. 4

LXXVI

AEROTITIS MEDIA

A CRITICAL REVIEW

REED W. HYDE, CAPT. USAF (MC)

RANDOLPH FIELD, TEXAS

Aerotitis media has been a problem since 1783 when one of the early balloonists first reported severe pain in his right ear resulting from an ascent to 10,500 feet.⁹ About 100 years later considerable interest in the effects of pressure changes on the human body was stimulated in Europe with recognition of the occupational hazards of the caisson worker. In 1900 Heller, Mager, and van Schroetter³² published an extensive pioneer study of this subject with particular reference to the ear. During World War I, the altitudes and rates of descent attained by aircraft at that time were not sufficient to attract much attention to the problem of otitic barotrauma.

Armstrong and Heim in 1937³ first described the condition as a distinct clinical entity and introduced the term *aero-otitis media*. During World War II it was known by a variety of names, including, among others, *aviation pressure deafness* and *acute otitic barotrauma*.¹⁸ The term more commonly used in this country today is *aerotitis media*.^{2, 60, 63, 75}

The original definition of Armstrong and Heim³ has been subject to some criticism, but is a good one and is generally accepted: "An acute or chronic traumatic inflammation of the middle ear caused by a pressure differential between the air in the tympanic cavity and that of the surrounding atmosphere." It is considered to be one form of secretory otitis,³³ and must be differentiated from otitis media of infectious origin; rarely, infection is superimposed as a complication.

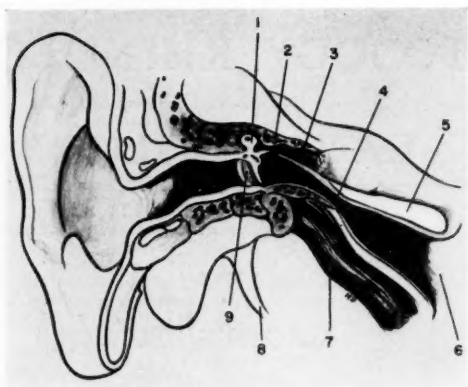


Fig. 1.—Longitudinal section through the eustachian tube; 1. head of malleus; 2. tympanic cavity; 3. tensor tympani muscle; 4. isthmus; 5. eustachian cartilage; 6. pharyngeal orifice; 7. levator veli palatini; 8. styloid process; 9. tympanic membrane.

Aerotitis may occur in caisson workers and submariners, and is very common in those who fly, whether in military or in commercial aviation. It is one of the three most common passenger discomforts in present-day commercial aircraft; airsickness and hypoxia are the other two.⁷¹ Pressure chambers, oxygen equipment, and jet aircraft have all contributed to the problem.

A considerable amount of literature upon the subject has accumulated in recent years, much of it based on research activities which grew out of World War II, but there are conflicting reports as to its incidence, the importance of various predisposing factors, complications, and methods of treatment. For these reasons and because the disease is becoming increasingly important to the otolaryngologist, a critical and systematic review of the literature is justified. It is felt that many otologists who have not had experience as flight surgeons may not be sufficiently informed as to the etiology, recognition, and treatment of this disease. Aerotitis will be seen increasingly often in private practice with the present popularity of commercial air transport, and particularly now that jet transport is an actuality.

Because the structure and function of the eustachian tube is of primary importance in explaining the effects of pressure change upon the middle ear, its anatomy and physiology will be briefly reviewed.

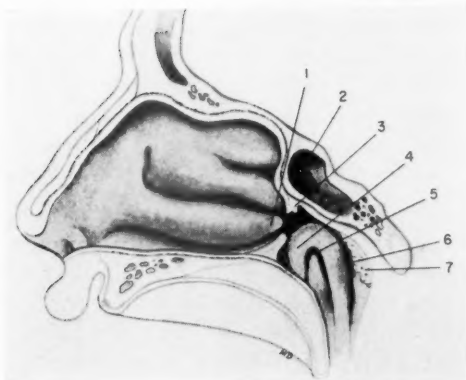


Fig. 2.—Relations of the tubal orifice; 1. posterior tip, inferior turbinate; 2. sphenoid sinus; 3. posterior end of nasal septum; 4. torus tubarius; 5. pharyngeal orifice; 6. fossa of Rosenmueller; 7. adenoid.

1. Anatomy of the eustachian tube

The eustachian tube extends backward, outward, and upward 31 to 38 mm from its pharyngeal orifice on the lateral wall of the nasopharynx to its tympanic orifice on the anterior wall of the middle ear (Figure 1). The medial two-thirds is cartilaginous, and joins the bony lateral one-third at an angle of approximately 160 degrees; at this point, the *isthmus*, the lumen is only 2 to 3 mm in height and 1 to 1.5 mm wide.²⁷ If the main axis were drawn through the tube it would go straight through the three main planes of the skull and form an angle of about 45 degrees with each of them.⁷⁷

Knowledge of the location and relations of the tubal orifice is important in nasopharyngoscopy and in attempting catheterization. The average distance of the orifice from the anterior nasal spine is 7.9 mm.²⁷ The distances from the posterior edge of the nasal septum, from the posterior end of the inferior turbinate, and from the vault of the nasopharynx are all close to 1 cm (Figure 2). The distance from the posterior pharyngeal wall with its adenoid growths averages 1.5 cm. At the pharyngeal orifice the axis of the lumen forms an angle of 130 degrees with the lateral wall of the nose:⁷⁷ thus the curved tip of an eustachian catheter should be bent at an angle of 130 degrees in order to have the tip of the catheter pointing exactly in the direction of the axis of the tube. This must be kept in mind so as to avoid reducing the effectiveness of catheterization and, more important, traumatizing the soft tissues.

The most prominent landmark on the lateral wall of the nasopharynx is the torus tubarius which bounds the pharyngeal orifice of the tube on three sides and is formed by the blunt medial end of the tubal cartilage and is covered by nasal mucous membrane. The fossa of Rosenmueller, lying between the torus and the posterior wall of the nasopharynx, is sometimes filled by lymphoid tissue or bridged by adhesions which may interfere with tubal function.

The tubal cartilage is in the form of an elongated triangular sheet with its apex at the isthmus and its base extending medially to the torus. On cross section near its mid-point it resembles a shepherd's crook, with a large posteromedial lamina and a smaller lateral lamina. The lateral and inferior walls of this portion of the tube are supported by a dense fibrous membrane, the salpingopharyngeal fascia.

The eustachian epithelium is continuous with that of the nasopharynx, being mostly pseudostratified ciliated columnar. Lymphoid tissue is often found in the tunica propria, and may collect in follicles toward the pharyngeal end to form Gerlach's tubal tonsil. The amount of lymphoid tissue varies considerably from person to person, from one portion of the tube to another, and in the same individual from one time to another.^{27, 54} Whether or not this lymphoid tissue is normal or is associated with previous or present disease is open to question; the important point is that such tissue does exist in many individuals. The mucosa is richly vascular, which is important in fliers with upper respiratory infections; the resultant engorgement and swelling may interfere markedly with tubal function.

In attempting to elucidate the isolated function of individual tubal muscles attention should be drawn to the interrelation of the muscles of the eustachian tube, soft palate, and pharynx.⁷⁷ Recent literature gives more convincing descriptions of the anatomy and function of these muscles than do the standard textbooks. The tensor veli palatini dilates the lumen of the tube; the levator veli palatini functions chiefly to elevate the soft palate, but may also open the pharyngeal orifice. The salpingopharyngeus forms a fold which is the downward continuation of the posterior lip of the torus; it may dilate the tubal orifice slightly. The superior constrictor and the pterygoid muscles have also been claimed to play some role in tubal function.^{15, 27, 42, 50, 58, 64, 77}

Perlman has published excellent slow-motion pictures of the mouth of the eustachian tube.⁵² At rest the orifice appears as a vertical crescentic slit with the anterior and posterior lips of the torus not quite in contact and often bridged over by a thin film of mucous. In

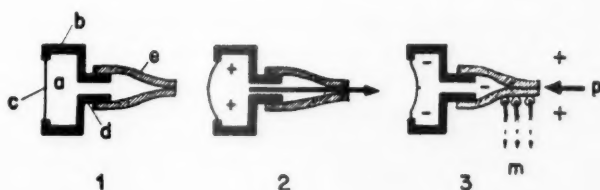


Fig. 3.—Diagram (after Frenzel) showing pressures in the tympanic cavity and the action of the eustachian tube during flight; a. tympanic cavity; b. bony capsule of tympanic cavity; c. tympanic membrane; d. bony portion of eustachian tube; e. cartilaginous part of the tube which is normally closed; m. muscular tension used to open the tube; p. compressed air in the nasopharynx; 1. normal condition; 2. ascent (decompression); 3. descent (recompression).

a. Normal Function

phonation the anterior lip remains stationary while the posterior lip appears to be lifted passively backward, upward, and medially as the soft palate and the floor of the tube are elevated. In swallowing, the two lips move actively in opposite directions so that the opening is greater, deeper, and rounder than on phonation. Not infrequently scarring and adhesions distort the picture to a variable degree.

2. Physiology of the Eustachian Tube

The most important function is ventilation of the middle ear, whereby the pressure on the two sides of the tympanic membrane are equalized. Other functions include drainage of accumulated fluids from the middle ear and closing off the tympanic cavity from the pharynx to prevent the fatiguing effects of autophony (in which one's own voice and respiratory sounds are heard loudly in the affected ear, occurring in the presence of a persistently patent tube⁵⁰).

The cartilaginous portion of the tube is normally closed when at rest, and is actively opened by yawning, sneezing, and swallowing (but not by every act of swallowing).⁵¹ In addition to this active opening of the eustachian tube, there is a *passive* opening whereby air can move through the tube toward the nasopharynx. It must be remembered that, except for the tympanic membrane, the middle ear and the mastoid air cells form a rigid-walled structure which communicates with the exterior only through the eustachian tube. The tube appears to act as a flutter valve between the pharynx and the middle ear (Figure 3); this allows the exit of air from the middle ear more readily than its admission from the nasopharynx.^{3, 24, 45}

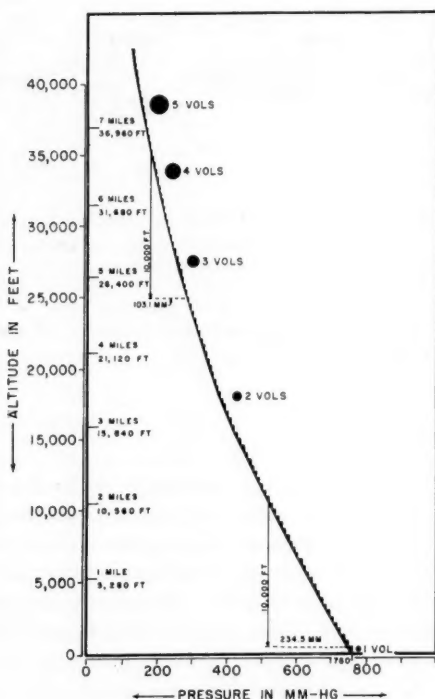


Fig. 4.—Pressure-altitude curve. Equal increments of altitude involve greater changes in pressure at low altitudes than at high altitudes. Changes in the volume of a gas have an inverse relation to changes in pressure; alterations in temperature are not considered in this graph.

b. Barometric Function

(1) *Ascent (decompression)*. If one ascends from sea level without swallowing, the gradually decreasing atmospheric pressure causes a slight sensation of fullness in the middle ear at an altitude of 110 to 150 feet.³ This sensation is accompanied by slight bulging of the tympanic membrane and slight conductive deafness. As ascent continues these changes increase until at about 500 feet a “click” is heard and the tympanic membrane returns to its normal, or nearly normal position. These changes result from the forcing open of the eustachian tube by the excess pressure in the middle ear and indicate that this excess pressure has been relieved by a sudden escape of air from the middle ear to the nasopharynx.

This cycle is repeated as ascent continues. With ascent equal changes of pressure involve increasing intervals of altitude, resulting in a nonlinear pressure-altitude curve (Figure 4). In spite of this, the eustachian tube is said to open at more or less equal altitude intervals. The combination of passive tubal opening and the normal action of swallowing allows one to ascend to altitude with little or no difficulty, even with moderate impairment of eustachian tube function. With rates of decompression higher than 120 mm Hg a minute, according to Chang's group,¹⁴ these periodic openings and closures of the tube no longer occur, at least in monkeys; instead there is a sustained patency of the tube which continues as long as the rate of pressure development in the middle ear exceeds the rate of outflow of air through the tube. This has been confirmed in the low-pressure chamber; Luft⁴¹ has been experimentally explosively decompressed from sea level to 50,000 feet in 1.0 second on numerous occasions, and reports no appreciable effect on his ears.

(2) *Descent (recompression)*. Whereas ventilation of the middle ear may be entirely passive during ascent, a completely different mechanism is involved on descent, and most cases of aerotitis media occur at this time.^{11, 18, 67, 76} This fact is explained by the "flutter valve" action of the eustachian tube. Because of negative pressure in the middle ear the normal closure of the tube becomes even tighter, and the tube must be opened by muscular action or by therapeutic inflation. If a negative pressure of 80 to 90 mm Hg is allowed to develop in the middle ear, "locking" of the tube occurs and it becomes impossible for the individual to overcome this force and open the tube without assistance.³

The pressure differential built up in the tympanum depends not only upon the amount of descent but also upon the altitude at which this descent occurs (Figure 4). Locking occurs much more readily at lower altitudes and can be readily overcome by immediate return to a higher level. It is also more likely to develop as the rate of recompression is increased.

(3) *Methods of autoinflation*. Yawning and swallowing are the most frequently used methods of ventilating the middle ear in flight. Although the tube is opened briefly during the swallowing, it can be kept open for a longer period of time (at least many seconds) by means of the Valsalva maneuver. Most experienced fliers are familiar with this maneuver, which has the disadvantage of requiring the use of one hand to close the nose. Resistance of the tube to this maneuver is greatly increased by assuming the prone position and flexing the neck toward the chest.⁵¹

Another method of "clearing the ears" used by many fliers is a grinding movement of the lower jaw. In a series of 63,000 low-pressure chamber "runs" to altitude in the Royal Canadian Air Force, those individuals who had difficulty clearing their ears often helped considerably when they were encouraged to relax rather than "clench their jaws."⁶⁷ This would be an argument for the passive effect of the pterygoid muscles on eustachian tube function.

The *pharyngeal pressure test* of Frenzel²⁴ is an even more effective means of opening the closed tube. With the nostrils and glottis closed, the air in the pharynx is compressed by the muscles of the floor of the mouth and tongue. It might be described as the action performed directly preceding a forceful "clearing of the throat;" as with the Valsalva maneuver, one can maintain patency of the tube for many seconds. Not only are the positive pressures obtained by this method higher than with the Valsalva maneuver, but, more important, the tube is opened at a much lower pressure. Ohnacker (cited by Frenzel) described a case in which the Valsalva maneuver could not open the eustachian tube with a pressure of 90 mm Hg, whereas it opened at 20 mm Hg using the pharyngeal pressure test. This method may be somewhat more difficult to teach the novice, but these authors advise that it be taught any flier having difficulty clearing his ears. Luft⁴¹ has used this method himself for many years in pressure chamber experiments, and has frequently "come down" from 50,000 feet to sea level in 12 seconds with no ear complaints.

c. Tests of Eustachian Function

In the selection of aviation cadets and in the examination of anyone suffering repeated attacks of aerotitis media, it is of considerable importance to determine the functional ability of the eustachian tube.

(1) *Toynbee's maneuver.* The tympanic membrane is observed while the tube is exposed to the small pressure change developed within the pharynx during the act of swallowing with the mouth and nose closed. If the drumhead is seen to move, normal tubal function can be assumed. This simple test has the advantage of examining the tube under normal physiological conditions, i.e. during swallowing.

(2) *Valsalva's maneuver.* With the mouth and nose closed, forceful expiration through the nose is attempted. Successful middle-ear ventilation may be verified objectively by auscultation or by direct visualization of the tympanic membrane. The latter method is said to be more accurate.¹⁸ Gidoll²⁶ describes a right-angle oto-

scope by means of which the examiner can view the drumhead while, at the same instant, via an auscultation tube, he can distinguish the character of the sounds coming through.

Failure to perform the Valsalva maneuver on the ground does not necessarily imply that an individual will be unable to autoinflate successfully in flight, and should not by itself disqualify an applicant for flying training. On the other hand, many people who perform this maneuver beautifully in the examining chair subsequently develop aerotitis media. Possible causes for these apparent discrepancies will be discussed later.

(3) *The pneumophone.* Indirect evidence of a pressure difference on the two sides of the tympanic membrane can be obtained by use of the pneumophone.⁷³ A small hand bulb and a source of sound are connected with a tube sealed into the external auditory meatus of the subject by an ear tip. A low frequency tone of constant intensity presented to that ear through the tube will be at optimal loudness when the pressures on both sides of the drumhead are equal. When middle-ear pressure is reduced, the subject hears the sound loudest when the examiner reduces the pressure in the external ear sufficiently to equalize that in the middle ear. Thus the examiner can determine the presence of a negative pressure in the middle ear and obtain indirect evidence of tubal insufficiency.

(4) *Inflation.* Inflation of each tube separately can be done through a catheter, or of both tubes together by means of a Politzer bag or some other source of pressure. Politzer's method is used most commonly but does not give accurate measurement of the degree of tubal adequacy. Perlman⁵¹ has developed a method for quantitative evaluation of tubal function by means of which the middle ear may be inflated using just the amount of pressure necessary rather than the uncontrolled excessive pressure introduced by the Politzer bag. Campbell¹⁰ and Ogden⁴⁸ utilized a modification of this method in their work on the problem of aerotitis during World War II.

3. Etiology of Aerotitis Media

It is generally agreed that aerotitis media occurs as a direct result of failure to equalize differences in pressure between the middle ear and the atmosphere. Thus it is dependent upon eustachian tube function and occurs subsequent to failure to open the tube voluntarily or inability to open it when exposed to changes in barometric pressure. Increasing density of air encountered on descent and rate of descent are important factors; a given change in altitude near sea level entails larger pressure differentials than at high altitudes

(Figure 4). Duration of the action of negative pressure in the middle ear is important.¹⁴ The low temperatures existing at high altitudes do not affect the incidence of aerotitis.³⁷

Absorption of oxygen from the middle-ear cavity also tends to produce or aggravate otitic barotrauma. This is most important in explaining "delayed" or "secondary" aerotitis media occurring in fliers who have been breathing oxygen and in whom the onset of symptoms is often delayed until the following morning or after an interval of sleep.^{6, 8, 11, 75} This type of aerotitis is being recognized more and more frequently today with the increased use of oxygen in high-altitude bombers and in jet aircraft.

Aschan⁴ suggests that these purely mechanical factors may not entirely explain the pathogenesis of aerotitis. In a series of carefully controlled animal experiments he has shown that both reduced and increased partial pressures of oxygen *at a total ambient pressure of one atmosphere* will produce vascular changes in the mucosa of the middle ear and of the nose and paranasal sinuses identical to those caused by changes in total barometric pressure. He did not get these changes to any appreciable degree with oxygen poisoning tests in a low-pressure chamber when the oxygen pressure was about 0.45 atmosphere. Since this latter situation is more nearly comparable to that of a flier breathing oxygen at altitude, where the total barometric pressure is reduced, it might be inferred that this particular factor is one of academic interest. This contention is supported by material in the field of physiology.¹ Toxic effects are practically never produced by breathing oxygen below a concentration of 60 percent of one atmosphere.^{5, 65}

There are situations where the mechanical factors described cannot apply. If a dry perforation of the tympanic membrane is associated with tubal obstruction, air would flow out of the middle ear during ascent and inward though the perforation during descent.¹¹ An ear that has had a radical mastoidectomy is not subject to aerotitis media. On the other hand, in an individual with an abnormally patent eustachian tube gases may flow readily back and forth through the tube during changes in pressure. These are extreme examples and obviously do not represent the average flier. A discussion of the various conditions which predispose to aerotitis media is now in order.

a. Failure to open a normal tube

An unconscious or sedated patient being transported by air, a sleeping passenger, and a markedly hypoxic flier might all have normal eustachian tubes and yet be unable to swallow as often as neces-

sary for adequate ventilation of the middle ear during changes in altitude. Carelessness, preoccupation, inattention and ignorance of the necessity for clearing the ears are etiologic factors. Inexperience is a most important cause of aerotitis media.^{18, 67, 68}

The value of preflight indoctrination, including the use of pressure chamber flights, is widely recognized in military aviation and cannot be overemphasized. Ferris and Engel show graphically a striking decrease in the incidence of inadequate middle-ear ventilation in 78 subjects during descent from seven consecutive high-altitude chamber flights.²⁵ Royal Canadian Air Force data⁶⁷ based on 63,000 low-pressure chamber "runs" show a progressive decrease in incidence on successive flights.

In his observations on the training of submarine personnel in the German Navy, Tonndorf⁶⁹ comments on the beneficial effects of training in the reduction of aerotitis media. Even in the schnorkel submarine, which involved considerably greater and more frequent changes in pressure, experienced submariners were able to sleep comfortably with unconscious equalization of these pressure changes after a period of adjustment varying from a few days to a few weeks. Zoellner⁷⁷ discusses the effect of training on strengthening the muscles of the eustachian tube. This was particularly beneficial in individuals in whom he found "insufficiency of the tubal openers" and an otherwise normal tube.

Even among experienced personnel, however, aerotitis is a problem. The fact is recognized that ignorance of and ineptitude in the performance of Valsalva's maneuver is common, and the "practice effect" following proper indoctrination is important in lowering the incidence of aerotitis media.¹⁸

b. *Permanent Eustachian Stenosis*

An occasional case of aerotitis may be due to permanent stenosis of the eustachian tube. Stewart et al⁶⁷ conclude that permanent eustachian obstruction is the cause of aerotitis media in only a small number of cases, but that these men should be eliminated from aircrew training as early as possible. A most important consideration in detecting these individuals is a history of repeated attacks of aerotitis while flying in the absence of any upper respiratory infection.

Faultily performed adenoidectomies and nasopharyngeal infections in childhood predispose to the formation of adhesions in the nasopharynx, some of which may interfere with tubal function. Lion³⁹ states that some of these patients with nasopharyngeal crypts and folds complain that they "cannot click their ears" on the af-

fected side and that they are easily susceptible to catarrhal (secretory) otitis or to aerotitis media.

Any condition in the eustachian tube predisposing to chronic inflammatory changes would predispose to cicatricial stenosis of the tube. Infection, allergy and trauma are the most frequent irritants within the tubal lumen.²¹ These same conditions in the nasopharynx may sometimes cause obstruction of the tubal orifice.

c. Temporary Eustachian Obstruction

Reliable criteria for the prediction of the occurrence of aerotitis media would be of considerable practical importance. Most of the literature on the subject emphasizes the role of upper respiratory infection as a predisposing factor. Among other possible etiologic factors are included: lymphoid tissue in the nasopharynx, nasal obstruction, malocclusion, allergy, and a history of previous ear, nose, and throat complaints or operations.

(1) *Upper respiratory infection.* In addition to the common cold any nonspecific acute or chronic infection of the nose, sinuses, and nasopharynx will be considered under the term *upper respiratory infection*. These conditions predispose to inflammatory swelling which may or may not involve the mucosa at the tubal orifice or in the lumen of the tube.

The relation between upper respiratory infection and aerotitis media has been frequently mentioned. Armstrong² has published a diagram suggesting a rough correlation between the incidence of upper respiratory diseases and aerotitis media during a three-month period. In one series of 100 cases of aerotitis,¹⁸ McGibbon noted 25 with infections of the upper respiratory tract, of which only 7 were classified as "colds;" the others included infections of the tonsils, adenoid, or maxillary sinus. Bateman¹⁸ concluded that the only important complication caused by flying with an active sinusitis is aerotitis media (otitic barotrauma); this occurred in 14 of 130 men who flew with sinusitis. Haines and Harris²⁹ state that "the presence of colds is of less significance in aerotitis media than is usually claimed."

Stewart and his co-workers⁶⁷ present data based on a total of nearly 15,000 men over a twelve-month period which do not show any correlation between the incidence of otitic barotrauma and upper respiratory infection. They agree that some relation does exist and that increased difficulty may be experienced in ventilating the middle ear in the presence of an upper respiratory infection, but they state that the importance of this relationship has been overem-

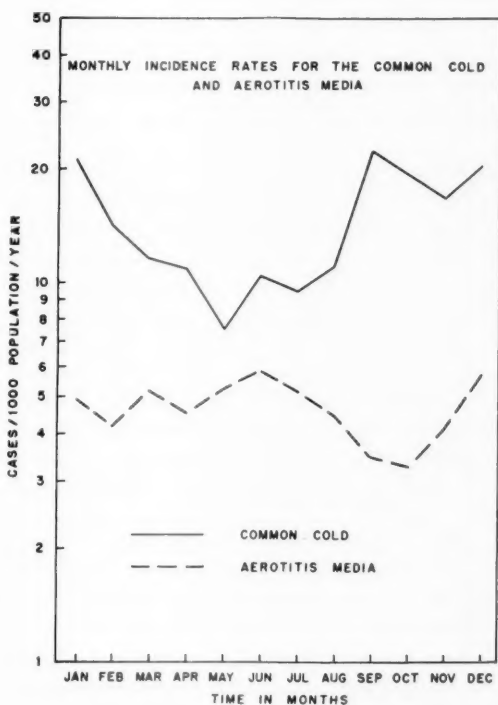


Fig. 5.—Monthly incidence rates for the common cold and for aerotitis media. All rates are expressed as cases per 1,000 population per year.

phasized. They feel that the grounding of all airmen suffering from coryza or pharyngitis would result in an unnecessarily high number of noneffective personnel. According to these authors, the decision as to whether or not a man may fly should depend upon his ability to ventilate his ears adequately rather than upon the presence of an upper respiratory infection.

Recent data from the U. S. Air Force,⁴³ based on a total of approximately 10,000 man years of exposure to aerotitis media, fail to show the direct correlation between the incidence of aerotitis and that of "the common cold" suggested by other studies (Figure 5). The study from which these figures were derived was of several months' duration, representative of all seasons of the year; it represents the findings of several different flight surgeons, but can be

accepted as being of valid statistical scope.* It is possible that by removing fliers with head colds from flying duties the flight surgeon is removing them from the risk of aerotitis media, thereby altering the rates for this condition and altering the actual relationship between aerotitis and the common cold.

(2) *Lymphoid tissue.* The role of infection cannot be separated from that of lymphoid tissue in obstruction of the eustachian tube. In the normal nasopharynx and fossa of Rosenmueller lymphoid tissue is often seen, but within the lumen of the eustachian tube it is said by some^{20, 21} to occur only with associated pathology. On the other hand, there are those⁷⁸ who feel that there is a diffuse layer of lymphoid tissue beneath the epithelium throughout the length of the tube. Lymphoid nodules have been observed in all parts of the tube in individuals with normal hearing.⁵⁴ Tubal obstruction may occur in any part of its length, but in actual practice it appears to occur most commonly at the pharyngeal end. Quiescent adenoid tissue, often present in adults, need not necessarily obstruct the eustachian tube orifice. It is, however, subject to inflammation and may on occasion be responsible for aerotitis media occurring for the first time in an individual who has had considerable flying experience.⁷⁶ When the inflammation subsides such an individual may have no further difficulty.

Shilling et al⁶³ found a positive correlation between the incidence of aerotitis media and both the size of the adenoid and the presence of lymphoid tissue around the tubal orifice; however, "The magnitude of the relationship did not permit good prediction in individual cases." Many of their subjects with perfectly open tubes developed aerotitis while many with heavily covered tubes did not. Stewart and his group⁶⁷ do not mention lymphoid tissue as an etiological agent in their comprehensive study of the subject.

In the U. S. Army Air Forces an Aerotitis Control Program⁷⁸ was begun in May 1944 when a group of otologists was called together to discuss the problem of aerotitis and the irradiation treat-

*Incidence rates were calculated by a standard procedure as follows:

$$r_{adj} = \frac{C_i}{P_t} K \quad \text{wherein}$$

r_{adj} = specific disease rate as the number of cases per 1,000 population per year.

C_i = cases of the specific disease per reporting period.

t = time of exposure in the units desired.

P = average population exposed to the risk of disease.

$K = 1,000$.

ment of hyperplastic lymphoid tissue about the eustachian tube orifice as initiated by Crowe.¹⁷ Control studies, unfortunately, were limited because of the acute need for treating fliers who had difficulty in clearing their ears.

A question was raised in connection with this program some time after World War II:⁷⁹ "In 1944, at Westover Field, Mass., where irradiation therapy was used extensively, I examined 1,000 soldiers routinely with the nasopharyngoscope. By the greatest stretch of the imagination I found 7 percent with hypertrophied lymphoid tissue involving the eustachian tubes. At the same field 37 percent of all fliers were treated by irradiation. Question: Were 90 percent of these soldiers treated needlessly or were my 7 percent observations wrong?"

As regards the pharyngeal tonsil, the impression gained from the literature is that enlarged tonsils per se are not an important factor predisposing to aerotitis. If they are chronically infected they may be a factor.¹⁸ In general it is not so much the amount of hypertrophied lymphoid tissue that matters but rather its location, particularly with respect to the eustachian tube. Pittmann⁵³ states that embedded hypertrophied tonsils may cause a pathologically patent tube. The lateral pharyngeal band or curtain of lymphoid tissue behind the posterior pillar occasionally extends upward so as to obstruct the eustachian orifice.²⁸ Fowler²² points out that hypertrophied tonsils merely indicate that lymphoid hyperplasia may also be present in the nasopharynx, and that any improvement sometimes noted subsequent to tonsillectomy is probably most often due to improvement of a chronic infection in the upper respiratory tract.

(3) *Nasal obstruction and allergy.* Nasal obstruction unassociated with any infectious process is often mentioned as a possible etiological factor in aerotitis media. Septal deviations or spurs, nasal polyps, polypoidal posterior tips of turbinates, foreign bodies and tumors can all obstruct nasal breathing. Wright⁷⁶ sent over 100 subjects with marked septal deviations into the low pressure chamber and found they developed neither aerotitis media nor aerosinusitis. Teed⁶⁸ believes that selective physical examinations should lay more stress on patency of the eustachian tubes and less on septal deviations.

Allergy is considered by many to be a predisposing factor, causing eustachian obstruction either by edema of the tubal mucosa or by an enlarged posterior turbinate tip overlying the eustachian orifice.^{11, 21, 35, 68} Lymphoid hyperplasia is frequently observed in individuals with nasal allergy. Dickson et al¹⁸ found that subjects

complaining of recurrent nasal "catarrh" were more prone to develop middle ear difficulties than those with no such history. No controlled studies of the isolated effects of allergy in producing aerotitis were found in this review. Recent clinical observations³⁵ suggest that allergy is much more commonly observed in people with repeated complaints of tubal closure during flying than has previously been stressed. Simple eustachian tube closure may be a fairly common manifestation of allergy. Allergic survey and treatment will be more effective than repeated tubal inflations in solving this problem. Recurrent allergy of the tubal or nasal mucosa may explain some cases of aerotitis occurring in individuals whose tubal function tests are normal in the examining chair.

(4) *Malocclusion.* Costen¹⁵ demonstrated anatomically that the membranous anterior wall of the cartilaginous portion of the eustachian tube could be compressed by the relaxed soft tissues in cases of overclosure of the lower jaw. Others believe the mechanism of obstruction in these cases may be by means of lymphatic stasis secondary to insufficient muscular activity. Willhelmy⁷⁴ applied the concept of malocclusion to aviation medicine. There are varying opinions as to the importance of this factor in the etiology of aerotitis media,^{18, 22, 63, 67, 74} but the general impression is that it does play a small but definite role in tubal obstruction. Where indicated, corrective dental measures have given excellent results.⁶³

(5) *Other factors.* Dickson's group¹⁸ felt that previous aural pathology might be a contributory factor, but that scarring of the tympanic membrane probably did not by itself predispose to acute otitic barotrauma. Fowler²² placed previous otitis media of any type, acute or chronic, fourth in importance in a list of fourteen predisposing and contributing factors.

In their study of submarine trainees, Shilling and his co-workers⁶³ found that efforts at predicting the occurrence of aerotitis media by examination were generally unsatisfactory; however, the chances were very high that an individual suffering one attack of aerotitis would again have difficulty on subsequent exposure to high-pressure air (50 pounds). A recent attack of aerotitis media will predispose to further attacks if the subject is again exposed to pressure changes before the middle-ear tissues have returned to normal.

Cleft palate and paralysis of the soft palate are mentioned as factors in tubal obstruction, but are not often seen in military aviation except in air evacuation flights. Theoretically, a tracheotomized patient might have trouble clearing his ears by means of the Valsalva

I	FIGHTER AIRCRAFT	4.0
II	LIGHT BOMBARDMENT AIRCRAFT	7.3
III	TROOP CARRIER AIRCRAFT	4.9
IV	MEDIUM BOMBARDMENT AIRCRAFT	6.9
V	ADMINISTRATIVE AIRCRAFT	1.9

Fig. 6.—Incidence rates of aerotitis media in flying personnel from five different plane types in the U. S. Air Force. All rates are expressed as cases per 1,000 population per year.

maneuver unless the tube were momentarily occluded during the performance of this maneuver.

Fowler²³ raises the possibility of polypoidal flaps or partially organizing exudate in the middle ear acting as a ball-valve to prevent air escaping from the middle ear and thus giving rise to symptoms on ascent. Other observers^{11, 18, 67} have commented on the rare occurrence of aerotitis developing during ascent. The possibility of mucous plugs obstructing either the tubal orifice or lumen is occasionally mentioned.

4. Incidence

Of 4,000,000 passengers carried by a commercial airline,⁷¹ only one per 2,000 passengers were recorded on the "passenger discomfort report" as having had "ear trouble." Wright⁷⁶ recorded an incidence of 6 percent in 150,000 routine low pressure chamber flights. Dickson and his group¹⁸ recorded a disability of 8.9 percent of 1,000 aircrew cadets in the low pressure chamber; another 4.8 percent had mild symptoms and injected drumheads relieved at once by autoinflation, and a further 27.7 percent showed injected drumheads but no symptoms. In one series of 708 trainees, Teed⁶⁸ found 30 percent with objective evidence of salpingotympanitis, although only 6.6 percent had complained of pain and been classed as failures.

The incidence of aerotitis found recently⁴³ in a large group of personnel on flying status flying in five different plane types of the U. S. Air Force is shown in Figure 6. These cases were all severe enough to cause removal from flying duties for a variable period of

time, and do not include those cases which were so mild that no loss of time was reported. It is not the purpose of this paper to discuss the variation in incidence among these different types of aircraft, interesting though it might be; the figures are presented merely to further emphasize that aerotitis is an important problem in aviation medicine and in otolaryngology today.

5. *Clinical and Laboratory Findings*

Ear pain has often been used as a rough criterion of the presence of aerotitis media in pressure chamber tests. It is not, however, a reliable index. Haines and Harris²⁰ observed that in 50-pound pressure chamber tests 12 percent of all ears pained enough that men reported it, although more than 25 percent developed some degree of demonstrable otopathy.

a. *Symptoms*

Aerotitis media may be unilateral or bilateral. The numbers complaining of monaural and of binaural symptoms are about equal, although objective findings are often observed in asymptomatic ears.^{18,30,57} In one group of 100 cases¹⁸ the predominant symptoms were: deafness alone, 31; deafness and pain, 55; deafness and tinnitus, 5; deafness and vertigo, 3; and pain alone, 6.

Pain varies from mild to almost unbearable, and its onset may be gradual or sudden. Most often the pain develops during descent, rarely on ascent; it sometimes occurs several hours after return to ground level. No matter how severe it may be, it is usually relieved upon ventilation of the middle ear. The severity of the pain does not necessarily parallel the objective findings; marked pain with no changes in the middle ear has been observed,⁶⁸ and slight or even no pain has been recorded with rupture of the tympanic membrane.¹⁸

Deafness is the most frequent complaint, but is often overshadowed by pain.¹⁸ Conductive in type, it is usually only slight and often transient, persisting from a few hours to many days.^{11, 22, 36, 63} Rarely a perceptive-type deafness may occur which is permanent and presumably due to labyrinthine hemorrhage.²² The more commonly observed conductive loss is usually reversible and is considered to be due to mechanical splinting of the ossicles or to edema and congestion of the mucous membrane of the middle ear.³⁶ It should not be confused with the acoustic trauma frequently found in flying personnel and associated with prolonged exposure to noise.

Some degree of *stuffedness* in the ears is almost always present, although for a variable period of time; this sensation is frequently

accompanied by a feeling of fluid in the involved ear. Wright⁷⁶ states that fluid appears sooner or later in the middle ear in almost every case of aerotitis media.

Tinnitus occurs not infrequently, but is not usually a predominant symptom. *Vertigo* is less common and is said to occur more often when the aerotitis is unilateral. In a series of 135 cases of vertigo due to obstruction of the eustachian tube, Merica⁴⁴ observed that the obstruction was unilateral in the majority.

In the delayed type of aerotitis the signs and symptoms are the same except for the delayed onset. This condition has been observed by pressure chamber workers as well as following actual flight, and seems to be definitely associated with the use of oxygen.^{6, 8, 11, 75}

b. Signs

The appearance of the drumhead often parallels the symptoms fairly closely but not always. Otoscopy is the most useful single diagnostic aid. Another common finding is a conductive type hearing loss. Examination of the upper air passages with particular reference to the nasopharynx and to eustachian tube function should be routine in all cases.

(1) *Tympanic membrane.* Otoscopy reveals changes varying from slight retraction with injection of the pars flaccida and along the handle of the malleus to hemorrhage in the middle ear with or without traumatic perforation. Various systems for classifying these changes appear in the literature;^{29, 68, 75} color drawings¹⁸ and color photographs^{2, 31} have also been published. According to Teed,⁶⁸ a grade 1 ear shows retraction with redness of Shrapnell's membrane and along the handle of the malleus; grade 2, retraction with redness of the entire drumhead; grade 3, the same plus evidence of fluid in the middle ear; and grade 4, hemotympanum and/or perforation.

Hemorrhage may occur within the layers of the tympanic membrane. This may be difficult to detect at first when the entire drumhead is red; it becomes more evident the second day when the erythema has subsided and the hemorrhagic area darkens. At this same stage the short process and handle of the malleus may take on a chalky white appearance, standing out in sharp contrast to the colored drumhead and enhancing the impression of retraction. One of the last signs of trauma to disappear is often a dark, dusky red streak along the manubrium.

Fluid occurs frequently but may be concealed by congestion, edema, or hemorrhage involving the tympanic membrane. If the

middle ear is completely filled with fluid it is more difficult to recognize; in these cases the entire drumhead has an amber color which may vary from a faint yellowish tinge to a dark blue, similar to the appearance of a hemotympanum.³³ The fluid may disappear within a few hours or not for several days; blood may persist for a week. Because of the direct communication between the mastoid air cells and the middle ear, the presence of fluid in these cells must also be considered in many cases.

Traumatic perforation due to barotrauma may occur in the anterior or posterior segment of the pars tensa, depending partially upon the previous condition of that particular membrane. Scar tissue areas on the drumhead may rupture on descent with relief of symptoms and no pain.⁷⁶ Stewart⁶⁷ felt that perforations of this sort rarely occur in an individual with normal eardrums. In his series of 24,000 airmen there were only five perforations, and in each case there was evidence of an old scar or calcareous plaque.

(2) *Nasopharynx*. The occasional appearance of fluid or blood at the eustachian orifice is the only finding in the nasopharynx which can be considered the result of aerotitis media.

A composite picture of the nasopharynx has been described by Wright;⁷⁶ he has observed these findings to be present almost constantly. They include: inflammatory swelling of the torus tubarius; adenoid tissue in the vault extending laterally to impinge on the torus; and edematous polypoidal posterior tips of the inferior turbinates. This picture has been described as being a factor in eustachian catarrh and responsible for considerable deafness in children. It is very often seen in nasal allergy, the role of which in predisposing to aerotitis has already been mentioned.

(3) *Hearing*. Acute aerotitis media is commonly accompanied by a temporary conductive deafness, lasting from an hour or two to two or three weeks.^{11, 18, 22, 36} The duration depends upon the magnitude of the initial trauma and the extent of secondary tissue damage. There appears to be no adequate correlation between the findings by otoscopy and the anticipated duration of the hearing loss. The loss may be from 10 to 30 decibels and is generally said to involve the low tones (128 to 2048 cycles per second).^{11, 18} Kos,³⁶ however, found that the greatest number of losses involved the higher frequency range (up to 11,584 cycles per second) and, further, that the most severe losses occurred in the higher frequencies. Shilling's group⁶³ found the most severe losses in those ears in which the tympanum was filled with free fluid, (20 to 30 decibels through

the frequencies 256 to 8192). Tuning fork tests reveal the usual findings associated with a mild-to-moderate conductive loss.

Experimental work has been done on the effect of pressure changes on auditory acuity.^{34, 40, 72, 73} Loch⁴⁰ found that both positive and negative pressure in the middle ear, when of marked degree, impaired thresholds for the frequencies from 32 to 1024 by as much as 20 to 30 decibels, and for the frequencies 2048 and 2896 by 10 to 15 decibels. There was very little effect for the frequencies 4096 and 5793. The results obtained by van Dishoeck⁷² with his pneumophone are much the same. Wever, Bray, and Lawrence⁷³ concluded from their animal work that the principal effect of these pressure changes on hearing is upon the ear drum, although minor effects may also arise in parts of the inner ear.

Different conditions of experimental material may explain the apparent discrepancies in these findings. Loch, Wever, and van Dishoeck experimented with alterations in atmospheric pressure not involving tissue changes, while in the material evaluated by Kos and Shilling actual tissue changes had taken place. In considering the transmission of sound within the middle ear the law of acoustic impedance may be applied as advocated by Campbell.¹² Changes in stiffness as may occur in simple alterations of pressure tend to involve the low frequency range only, while changes in mass as occur in the tissue alterations accompanying actual aerotitis media impair conductance for high frequencies.

(4) *X-ray findings.* Radiological investigations of the problem of aerotitis media have been done.¹⁸ McGibbon and Allen concluded that radiological examination of the mastoid was of neither diagnostic nor prognostic value in individual patients. They note, however, that when clouding of the mastoid cells does occur in association with aerotitis it requires a minimum of four weeks for complete resolution.

Rees-Jones and McGibbon⁵⁶ demonstrated obstruction of the eustachian tube in 36 patients with aerotitis media (aviation pressure deafness) by instilling a radio-opaque substance. In spite of the evident success achieved by these investigators, radiological examination would not appear warranted as a routine procedure; the diagnosis of acute aerotitis media is usually readily made on a basis of the history and otoscopic findings.

c. Pathology

For obvious reasons, pathologic studies of acute aerotitis media in human subjects are conspicuous by their absence from available

literature. Since the clinical entity "aero-otitis media" was first described in 1937, much has been written on the subject, but the only controlled pathological material has been obtained by animal experimentation.^{4, 18} The pathologic changes in the middle ear are presumed to be primarily vascular in nature and probably occur in the following sequence; some degree of hyperemia is nearly always present in the mucosa of the middle ear. Mucosal edema and submucosal hemorrhage may occur singly or together; the hemorrhagic areas vary from tiny petechiae to large confluent areas. As the condition progresses a serous effusion appears; this may become serosanguineous or there may even be gross hemorrhage into the tympanic cavity. Bacteriologic studies have shown these aspirated fluids to be sterile.⁷⁰

In discussing otitis media with effusion, Hoople³³ describes two types of fluid which may fill the middle ear. One is a thin, serous transudate caused by negative pressure in the middle ear. The other is mucoid in character and is exudative, usually being mixed with the serous transudate. Both types of middle ear effusion may persist for weeks, months, or years, according to Hoople, without any pathogenic bacteria. Aerotitis media is more commonly associated with the serous type which is usually completely resolved within two or three weeks if promptly and adequately treated.

Fowler²² has examined the temporal bones of experienced aviators who had had many hours of flying time and thus, presumably, had been exposed to either clinical or subclinical attacks of aerotitis media. He noted excess fibrous tissue in the middle ear of 4 out of 26 specimens; these permanent changes may have been due to previous middle ear infection, although none of these men had been on sick call because of their ears, and all had passed the regular physical examination for flying. Many observers have noted clinically that fliers with considerable flying time have thickened tympanic membranes, presumably the result of repeated insults to the middle ear structures causing irreversible changes.

6. *Differential diagnosis*

External otitis (including aerotitis externa), acute suppurative otitis media, acute secretory otitis media (otitis media with effusion), and myringitis bullosa hemorrhagica must all be differentiated from acute aerotitis media. In each case a history of recent exposure to pressure changes, as in an aircraft, is more suggestive of aerotitis media. Although otoscopic findings may be misleading, a careful

history and inspection of the tympanic membrane will usually give the correct diagnosis.

7. *Complications and Prognosis*

a. *Permanent deafness*

Although deafness is one of the commonest symptoms of aerotitis media, it is generally slight and only temporary. However, mild acute aerotitis may become recurrent or chronic when a flier is exposed to repeated pressure changes before complete recovery from a previous acute attack. The drumhead becomes more retracted, dull, and thickened. In 1942 McGibbon¹⁸ applied the term "aviation pressure deafness" to this condition because of the frequency of deafness as a complaint. It must be emphasized that casual treatment of an initial lesion may allow progression of tissue changes with resultant irreparable damage to the middle ear. These are the ears which may develop a permanent hearing loss. Fortunately most of these fliers can be restored to full flying status although the recovery period may be protracted. Kos³⁶ states: "Hundreds of flying personnel who have experienced aerotitis media once or several times reveal no permanent hearing impairment in the speech range."

b. *Suppuration*

Suppuration is a very uncommon complication of aerotitis media in fliers. Wright⁷⁶ observed only 39 cases of suppuration in approximately 9,000 cases of aerotitis. Stewart⁶⁷ reported 6 cases of acute suppurative otitis media among 1,505 airmen who developed aerotitis media; he was not convinced that all of these 6 cases were due to barotrauma.

Requarth⁵⁷ observed 12 cases of suppuration subsequent to barotrauma in 400 caisson workers who developed aerotitis media. Among 6,000 submariners, Teed⁶⁸ saw no cases of otorrhea that could have been considered to be the result of barotrauma. He did observe one case of purulent otitis media which was believed to be secondary to the use of the Valsalva maneuver shortly after leaving the pressure chamber. He concluded that the incidence of only one case in more than 6,000 men minimizes the use of this maneuver as an etiologic factor. It is recognized by flight surgeons that although many airmen fly with an existing upper respiratory infection and must clear their ears by some method of autoinflation, very few ever develop acute suppurative otitis media which can be attributed to this cause.^{3, 67, 76} The value of the Valsalva maneuver in preventing aerotitis media

far outweighs the remote possibility of introducing infection into the middle ear from the nasopharynx.

Tonndorf⁶⁹ noted an incidence of almost 10 times as many cases of acute middle ear disease *of all types* among groups of 2,000 submarine trainees as among the 1,500 permanent party personnel at the same base. This was during the period from September 1942 to December 1943, and no statistics are available as to seasonal variation, incidence of upper respiratory infection, nor the various types of middle ear disease. His impression was, however, that there was a marked increase in the number of cases of suppuration among these recruits and, further, that barotrauma was definitely one of the causative factors. He concludes that men with subclinical acute aerotitis media subjected to repeated insults before restitution of middle ear structures to normal may be more susceptible to otitic complications than men with normal middle ears.

Conflicting reports as to the incidence of suppuration complicating aerotitis media may be due to at least in part to variations in the numbers and virulence of pathogenic organisms in a given area during a given period of time. This same factor may also help explain the variation in the figures correlating the incidence of aerotitis and upper respiratory infection noted earlier.

c. Prognosis

The prognosis in cases of acute aerotitis media is generally good, provided treatment is adequate and sufficient time is allowed for complete recovery; this varies from a few hours to three or four weeks. The rapidity with which treatment is initiated following exposure, as well as the type of treatment employed, may also affect prognosis.

8. Treatment

Treatment of aerotitis media may be divided into three phases: prevention, active therapy, and correction of contributing etiologic factors. Application of some of these measures is more appropriate to and more readily achieved in military than commercial aviation. Airline fliers are indoctrinated in the same manner as are military flying personnel; airline passengers, however, cannot, for obvious reasons, be subjected to the same discipline and scrutinization.

a. Preventive measures

Preventive measures include a variety of points ranging from adequate physical examination of applicants for flying training to

the pressurizing of aircraft and control of rates of descent from altitude.

(1) *Physical examination for flying training.* Emphasis should be placed upon normal function of the eustachian tube rather than upon a deviated nasal septum or scarred tympanic membrane. When the otologic history is negative and the nasopharynx and eardrums appear normal, failure to perform the Valsalva maneuver does not prove tubal malfunction and should not disqualify for flying training. From the figures of Shilling and his group^{61, 62, 63} it appears that most of those who are "doubtful" in the performance of this maneuver will eventually "pass the pressure chamber test," but that special attention must be devoted to those who are demonstrably "poor." Those few individuals whose history and physical findings reveal permanent tubal stenosis should be eliminated from aircrew training.

An applicant with a head cold should be deferred temporarily. Subjects with chronic tonsillitis or sinusitis should have these conditions corrected unless they are mild or asymptomatic. If it is felt that correction of a deviated nasal septum will improve an existing associated infectious process, submucous resection should be recommended. More attention should be devoted to allergic manifestations than has been in the past. Active seasonal hay fever not responding to desensitization obviously should disqualify for flying training, but those individuals with a mild perennial type of vasomotor rhinitis are much more difficult to evaluate; a careful allergic history should be taken whenever indicated.

(2) *Indoctrination.* The importance to fliers of proper indoctrination in the act of autoinflation cannot be stressed too highly. This should begin with the initial physical examination for flying fitness; while testing the ability of an examinee to aerate the middle ear, a moment or two spent by the examiner in explaining the necessity for and the technique of the Valsalva maneuver to those applicants having any difficulty with it will be well worth the effort. In any unselected group of examinees from 20 to 30 percent may be unable to valsalva satisfactorily upon the first attempt.⁶⁵ Other methods of autoinflation which may be helpful in selected cases have already been described.

It is neither practical nor necessary to test every prospective flier in the altitude chamber as a selective measure. Once flying training has begun, however, at least one indoctrination chamber flight should be routine for all aircrew personnel. Special attention

should be given those men complaining of difficulty in clearing their ears, and any airman experiencing repeated attacks of aerotitis media should be thoroughly examined by an otolaryngologist.

Passengers in commercial aircraft should be given instructions with reference to clearing their ears. On sleeper planes it is advisable to awaken passengers during descent so as to avoid locking of the eustachian tube. The practice of feeding infants so as to make them swallow more frequently during descent is of help. There will always be some individuals unable to clear their ears because of varying degrees of temporary or permanent tubal stenosis.

(3) *Avoidance of flying.* It is impractical to advise grounding of every flier with an acute upper respiratory infection; there is evidence that such a procedure would not significantly reduce the incidence of aerotitis media anyway.^{67, 68} Proper indoctrination with particular reference to the necessity for middle-ear ventilation is of far greater value than overemphasizing the dangers of a head cold. Where possible, however, it would be better if a flier or prospective airline passenger with a cold could avoid flying for a few days or until his cold is improved; this is certainly true for those in whom adequate indoctrination and experience have not been achieved.

The ability to valsalva is a better criterion than the mere existence of acute coryza for acceptance for altitude chamber flights, according to experienced chamber observers. Stewart⁶⁷ noted that: "New medical officers began by excusing from tests large numbers of airmen who had an upper respiratory infection. After some experience they limited the group who were excused to much smaller numbers, without increasing, and sometimes actually decreasing, the incidence of otitic barotrauma."

Anyone who has had a recent acute attack of aerotitis should avoid flying until his middle-ear tissues have returned to normal. Depending upon the severity of the insult and the amount of pathology, this may require up to three or four weeks; occasionally an even longer period is necessary. The urgency of the situation requiring an individual to fly must be weighed against the possible complications and permanent middle-ear damage which may result from repeated attacks of acute aerotitis media. The very low incidence of suppuration as a complication has been mentioned; the incidence of persistent hearing impairment associated with chronic aerotitis is somewhat higher, although precise figures do not appear in the literature.

(4) *Vasoconstriction.* If persons with head colds must fly, they may possibly be better prepared to prevent middle-ear discomfort

by the prophylactic use of vasoconstrictors. The essential aim is the shrinkage of nasal mucous membranes, especially around the eustachian orifice. Individuals with allergic rhinitis should be cautioned against the prolonged use of nose drops; oral ephedrine might be substituted.

In separate studies of the effect of vasoconstrictors in preventing altitude chamber aerotitis media, both Shilling⁶³ and Ogden⁴⁷ found that no significant decreased incidence followed the use of these drugs immediately before flight. A beneficial result from the use of vasoconstrictors might possibly be due to the focusing of attention on the necessity for middle ear ventilation. The use of vasoconstrictors in treating upper respiratory infections and acute suppurative otitis media has been long recognized; their indiscriminate use in preventing repeated attacks of aerotitis media, however, cannot be condoned. Possibly more good might result from advising people who must fly with excessive nasal secretions to draw these secretions back into their pharynx during descent, rather than forcefully blow their nose and unnecessarily risk introduction of infection into the middle ear.

(5) *Pressurized aircraft.* The pressurization of modern aircraft has done a lot to reduce the incidence of aerotitis. In many of the present commercial aircraft, however, cabin pressure is maintained at 4,000 to 6,000 feet under normal cruising conditions, and may be higher when flying at a storm-evading level;⁷¹ the pressure differential between 8,000 feet and sea level (about 200 mm Hg) is still sufficient to cause considerable ear discomfort. In military aircraft, although explosive decompression offers but slight hazard to the middle ear, rapid recompression remains a very real potential cause of otitic barotrauma. For these reasons, an aircraft commander should always alert his crew before any descent or repressurization. Fighter pilots routinely "break pressure" many times, and must keep in mind the necessary adjustments to these sudden pressure changes.

(6) *Rate of descent.* Commercial aircraft normally descend from 300 to 500 feet per minute. Since the average person swallows involuntarily about once a minute, then, according to Armstrong,² descent at a rate of 200 feet per minute will usually cause no discomfort; 500 feet per minute, slight discomfort; and 1,000 feet per minute, moderate discomfort even though no voluntary effort is made to clear the ears. On the other hand, some pressure chamber workers routinely descend at extremely rapid rates—e.g., from 50,000 feet to sea level in 12 seconds.⁴¹ This enables them to "come down"

from altitude to sea level while employing only a single Valsalva or "pharyngeal pressure" maneuver. The vast majority of today's fighter aircraft, including jets, descend at rates between these two extremes, and are thus in the zone where the ears must be repeatedly ventilated if "locking" is to be avoided.

For routine pressure chamber flights the rate of descent is often measured in terms of millimeters of mercury per minute rather than feet per minute. The scale used at the U. S. Air Force School of Aviation Medicine, for example, is 27.1 mm Hg per minute.

(7) *Altitude factor.* A pressure differential can be built up in the middle ear much more rapidly at lower altitudes because of the slope of the pressure-altitude curve (Figure 4); a given change in altitude involves increasing differences in pressure as one descends towards sea level. This theoretical effect of altitude has been substantiated by clinical experience: many fliers, flight surgeons, and pressure chamber operators have observed that aerotitis media develops much more frequently at the lower altitudes. Most experienced fliers are aware of this fact and apply this knowledge by clearing their ears almost subconsciously as they descend.

(8) *Oxygen replacement.* For the delayed type of aerotitis media attributed to absorption of oxygen from the middle ear cavity, the breathing of helium-oxygen mixtures or of air rather than oxygen alone during descent has been suggested.^{24, 30, 66} Hall³⁰ found that the prophylactic inhalation of a helium-oxygen mixture (80-20 percent) failed to prevent painful ears and concluded that the administration of such mixtures was impractical. Post-flight inflation with helium has been used successfully in treating cases of aerotitis in fliers⁶⁶ as well as in caisson workers.⁵⁷

Bowen⁸ felt that "oxyotitis" could be prevented by introducing air with its normal concentration of almost 80 percent nitrogen into the middle ear during descent. If air were breathed instead of oxygen for the entire duration of descent, he found a definite decrease in the incidence of delayed aerotitis in the pressure chamber. This method might be useful under some conditions in the pressure chamber when rapid descents are possible, but in military aviation the danger of hypoxia is more important than the possible development of delayed aerotitis.

Since passengers in commercial aircraft are not normally required to breathe pure oxygen, theoretically they should not be subject to delayed aerotitis. Undoubtedly a few cases may occur from time to time, but not in appreciable numbers.

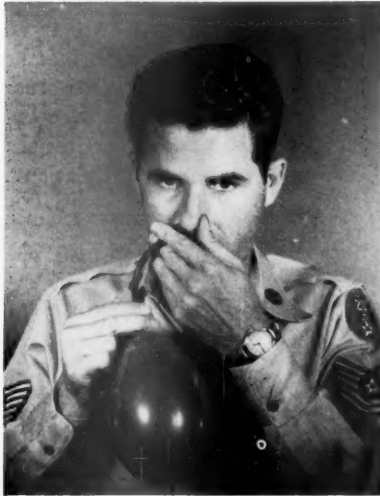


Fig. 7.—A simple instrument for ventilation of the middle ear (described by Fowler.²⁵)

b. *Active Therapy*

In the majority of cases of acute aerotitis media symptoms are mild, pain subsides early, and either no treatment or a minimum of conservative treatment is necessary. Resumption of flying should be avoided, when possible, until otoscope findings are negative and the physician is satisfied that recovery is complete. If a dark red streak is still visible parallel to the manubrium, or if the eustachian tube resists inflation, recovery is not complete and flying should not be resumed.

For more severe cases the primary concern is relief of pain which may occasionally be excruciating. Further objectives include: (1) relief of tubal obstruction, (2) neutralizing the vacuum effect, and (3) preventing recurrence of this vacuum effect.¹¹ It should be emphasized that the sooner treatment is begun, the sooner a cure is effected. An initial delay of more than two or three hours may make a difference of many days in the amount of time a flier is grounded.

(1) *Reascent.* Reascent in an aircraft or pressure chamber to the level at which successful aeration is achieved is the ideal treatment; this is followed by descent at a rate slow enough to allow the patient to keep his ears cleared until ground level is reached.

(2) *Vasoconstriction.* When the above-described method is not readily available, vasoconstriction followed by artificial inflation of the middle ear is the treatment of choice. Nasal secretions should be removed and then vasoconstriction may be accomplished by the use of nose drops or sprays, oral medication, or by the Proetz displacement technique.⁵⁵ Sufficient time to shrink the nasopharyngeal tissues must be allowed before attempting inflation.

A good description of the displacement technique as used by a flight surgeon in treating aerotitis media is given by Wiseheart.⁷⁵ His results were much better when treatment was initiated as early as possible after the onset of the disease.

(3) *Tubal inflation.* Resolution of acute aerotitis media begins when the pressure difference between the tympanic cavity and the outside air is neutralized. When vasoconstriction has been accomplished, the nose can be cleared again and then autoinflation is attempted. If the individual cannot clear his own ears, artificial measures must be employed. Inflation should not be attempted before full vasoconstriction has taken place.

(a) *Politzerization.* The Politzer bulb is widely used and is an item of supply for every flight surgeon. Fowler²³ used a modified version of the Politzer maneuver during World War II. He substituted an inflated toy balloon for the Politzer bulb and thus was able to deliver pressure over a longer period of time (Figure 7). Since there is never more than 30 mm of Hg of pressure developed during deflation of a toy balloon, no harm can come from this type of inflation, and it is often sufficient to overcome tubal resistance. Control of pressure is certainly more easily maintained this way than by energetically squeezing a Politzer bag.

Clinical observations by many flight surgeons confirm the impression that complications are very rarely introduced by these procedures, provided adequate precautions are taken and the method is not abused.

(b) *Controlled positive pressure.* In 1943 Perlman⁵¹ described a method for obtaining quantitative measurements of tubal function, utilizing a controlled source of positive pressure by means of which the middle ear can be inflated by just the amount of pressure needed instead of by uncontrolled excessive pressure. Ogden⁴⁸ found that the amount of pressure necessary to inflate an ear blocked by aerotitis media is variable but usually less than 46 mm Hg. In a few cases in which greater pressure was required no deleterious efforts were noted, but a pressure of 100 mm Hg was considered the upper limit

for safety. Following the principles described by Perlman, he used a simplified apparatus to deliver continuous controlled positive pressure to the nasopharynx in the successful treatment of a large series of cases of aerotitis media.

Using this continuous pressure technique, the ear can be successfully inflated with a minimum of pressure without the danger of overinflation and further trauma or even possible rupture of an already damaged tympanic membrane. No complicated apparatus is required for this procedure; any source of positive pressure in which the amount of pressure can be regulated will suffice. The control valve can be set initially to deliver 20 mm Hg pressure and the treatment begun. If this is not sufficient to relieve tubal obstruction the pressure can be raised by increments of 5 or 10 mm until inflation results. Success may be verified subjectively by the patient or objectively by auscultation, otoscopy, or both. On completion of treatment the patient is cautioned against indiscriminant attempts at autoinflation and too violent nose-blowing and should be told to return daily for observation as long as is considered necessary. If concomitant cold or nasopharyngeal infection is present, vasoconstriction and whatever other medication is indicated should be used at regular intervals. Occasionally the condition is serious enough to warrant hospitalization for a few days.

(3) *Eustachian catheterization.* Catheterization of the eustachian tube is frequently performed by otologists and occasionally by flight surgeons who are not otolaryngologists but who are obliged to treat cases of aerotitis media. A most important objection to catheterization is the possibility of trauma to the tissues at the eustachian orifice; Farrior²¹ stresses the point that trauma may result particularly if there is any inflammation of this delicate mucous membrane. He points out that cicatricial stenosis of the eustachian tube subsequent to trauma may result in permanent grounding of the flier concerned. Stewart and his co-workers⁶⁷ never had to use politzerization or catheterization to relieve an airman who had aerotitis media in their series of some 63,000 low-pressure chamber flights. It is a delicate procedure and should be done by trained hands only; in inexperienced hands far more damage can be done by the eustachian catheter than can be compensated for by successful inflation.

(4) *Relief of pain.* Analgesics may be necessary in severe cases, but rarely for more than 24 hours. The instillation of warm soothing oil or of warm water into the external canal has been found to give considerable relief from pain, but dry heat is safer, and is the form recommended by the Air Force School of Aviation Medi-

cine.^{7, 44} Rest and sedation are preferred by some to the use of ear drops.⁶⁸

(5) *Myringotomy.* Myringotomy has been considered as a form of treatment for aerotitis, but is not recommended for the flight surgeon untrained in otolaryngology. Canfield and Bateman¹³ in 1944 experimented with myringopuncture for relief of reduced intra-tympanic pressure in the altitude chamber. In 1949 Trowbridge⁷⁰ found that aspiration of the tympanum was most effective in relieving symptoms of acute aerotitis media, and, more important, shortened the average period of convalescence in his small series from 12 to 4 days. Seltzer,⁶⁰ in 1951, introduced a newly designed, double-barreled paracentesis needle for use in aerotitis media. The accessory barrel allows the passage of air in or out of the tympanic cavity when the fluid is withdrawn, thereby compensating for the fluid removed and avoiding the presence of a vacuum in the middle ear.

Withdrawal of the sterile fluid from the tympanum prevents its subsequent organization into adhesions which may result in permanent hearing impairment. Infection may usually be avoided by careful technique and institution of antibiotics in the few cases where their use is indicated. However, since more conservative measures of therapy have proven so universally satisfactory in the past, the use of myringotomy should be reserved for cases which do not respond to the usual methods of vasoconstriction and inflation.

c. Correction of Contributing Etiologic Factors

In any case of aerotitis media an effort should be made to evaluate possible etiologic factors with particular reference to the condition of the nasopharynx and a history of allergy. If lack of experience and inadequate indoctrination have been ruled out as factors, and if the individual develops repeated attacks when flying in the absence of acute upper respiratory infection, a thorough otolaryngological investigation is in order. Chronic infections of the upper air passages should be treated. Tonsillectomy and submucous resection have given disappointing results.^{68, 76} In those cases where surgery or irradiation of the nasopharynx or dental adjustment is definitely indicated, results have been gratifying.

(1) *Nasopharyngeal irradiation.* The use of radium or radon applicators in the treatment of lymphoid hyperplasia about the eustachian orifice proved satisfactory in the control of aerotitis media during World War II. Experience of the Army Air Forces Control Program showed it to be ideally suited for military use. It required

little time, interfered very little with combat or training schedules, and did not interrupt flying activities.⁷⁸ Because of the military situation existing at that time, no large scale control study was undertaken; in the United States men selected for treatment included many who had experienced no difficulty in clearing their ears. For these reasons, results are difficult to evaluate. Other flight surgeons have stated that irradiation is of value only in a small number of carefully selected cases.

Shilling and his group⁶³ concluded that *where indicated* radium therapy has a satisfactory effect on 90 percent of cases. They felt that x-ray therapy should also prove beneficial, but for administrative reasons were unable to evaluate its use in their study. Deep x-ray therapy was used recently by Dickson and McGibbon¹⁹ in the Royal Air Force on a series of very carefully selected cases of recurrent aerotitis; the conclusion was that: "Deep x-ray therapy is the only practical and safe method available at present to cause recession of lymphoid tissue situated *in the walls of the eustachian tube*." The authors feel that there is a definite field of usefulness for irradiation by deep x-ray in treating some cases of recurrent aerotitis media.

In selecting cases for treatment it might be well to consider these observations of Lindsay and Perlman:³⁸ "If no improvement in hearing can be demonstrated after tubal inflation it is unreasonable to attribute the loss to eustachian tube and reversible middle-ear disease and on that basis to initiate treatment of the eustachian tube to improve hearing." Again: "All degrees of lymphoid tissue hyperplasia may be discovered in the nasopharynx without evidence of ear disease and conversely a clean nasopharynx may be observed in the presence of middle-ear disease and eustachian tube obstruction." Certainly a careful, clean surgical adenoidectomy should have been performed and the possibility of allergy considered before irradiation is advised.

The technique of nasopharyngeal irradiation is adequately described elsewhere.^{17, 23, 78, 79}

(2) *Allergic Investigation.* The importance of allergy in the field of otology has been summarized recently by Jordan.³⁵ His observation that allergy is much more commonly associated with repeated complaints of tubal closure during flying than has previously been stressed deserves emphasis. Other otolaryngologists who have also served as flight surgeons^{11, 21, 22} have considered the possible role of allergy in predisposing to recurrent aerotitis media. The subject is included at this point to urge a more thorough appreciation of the necessity for a careful allergic history and complete

allergic investigation where possible and when indicated before advocating wholesale submucous resections and nasopharyngeal irradiation for the prevention and treatment of aerotitis media. Unfortunately, the undesirable side effects of available antihistaminics restrict their use for military flying personnel and thus further complicate the problem of allergy and aerotitis in fliers. The opinion of an allergist or of an otolaryngologist experienced in allergy should be obtained before instituting desensitization, elimination diets, or other procedures usually included in an allergic regime.

(3) *Dental therapy.* The impression gained from the literature on the subject is that malocclusion does play a small but important role in predisposing to tubal malfunction.^{16, 18, 29, 63, 67, 74} During World War II, Shilling's group⁶³ successfully applied dental therapy to 46 out of 50 men who had previously failed a 50-pound pressure test; a control series was included in this study. Costen^{15, 16} has had good results in treating two series of 400 cases of "mandibular joint syndrome," and Seaver⁵⁰ claims successful results with the use of a series of myotonic geniomandibular exercises. Unfortunately, detailed analysis of the results of treatment, particularly with reference to eustachian tube function, are not included in these latter studies.

In general, successful treatment appears costly and time-consuming, and should probably be reserved for those patients having repeated attacks of aerotitis media in whom all other measures have failed and in whom definite static or functional malocclusion or both can be demonstrated. Many cases have been seen in which malocclusion was present and no aerotitis ever developed. Costen¹⁶ gives a complete summary of the "mandibular joint syndrome" with details of treatment.

SUMMARY

Aerotitis media is defined as a barotraumatic lesion of the middle ear and its noninfectious nature is stressed. Infection is very uncommon as a sequella.

Incidence, etiology, and treatment are discussed, and new data are presented. Emphasis is placed upon the possible predisposing role of upper respiratory infection, nasopharyngeal lymphoid tissue, and allergy. Variations in season and climate and in virulence and frequency of organisms causing upper respiratory infections may partly explain apparent discrepancies in data.

A relation does exist between the incidence of upper respiratory infection and aerotitis media, but its importance has been overem-

phasized. Increased difficulty in the act of autoinflation may be experienced in the presence of a head cold, however, and as a rule it would be better if individuals with colds could avoid flying until the cold is improved.

More attention should be paid to the possible role of allergy in aerotitis media.

Ability to perform the Valsalva maneuver is a better criterion than the existence of a head cold or a deviated nasal septum for acceptance for altitude chamber flights. Ignorance of and ineptitude in the performance of the Valsalva maneuver is common, and the "practice effect" following proper indoctrination is important in reducing the number of cases. The value of the Valsalva maneuver in preventing locking of the eustachian tube with resultant aerotitis far outweighs the possible danger of causing suppurative otitis media by this method.

Ideal treatment is reascent to the level at which successful middle-ear ventilation is achieved; if this method is unavailable, vasoconstriction followed by middle-ear inflation is the treatment of choice. Dental measures and nasopharyngeal irradiation give good results in some cases of recurrent aerotitis, but the indication must exist.

Although conductive deafness is one of the commonest symptoms of aerotitis media, it is generally slight and only temporary. Sufficient time for complete recovery must be allowed before a patient who has had an acute attack of aerotitis is again permitted to fly. Casual treatment of an initial lesion may allow progression of tissue changes with resultant irreparable damage to the middle ear; these are the ears which may develop a permanent hearing loss. The number of fliers who are permanently grounded for aerotitis media appears to be very low.

Although all other etiologic factors theoretically may be controlled, there will always be cases due to the "human factor" (inattention, ignorance, etc).

ACKNOWLEDGEMENT

The author wishes to express his appreciation for advice and for invaluable assistance in organizing this material to Colonel P. A. Campbell, Lt. Col. J. E. Lett, Captain R. E. Johnson, Dr. J. Tonndorf and Dr. U. C. Luft, and for evaluation of statistical data to Captain W. W. Melvin. The author is also grateful to A/IC B. Ray for his assistance with the manuscript.

USAF SCHOOL OF AVIATION MEDICINE.

REFERENCES

1. Armstrong, H. G.: The Toxicity of Oxygen at Decreased Barometric Pressures, *Mil. Surgeon* 83:148-151, 1938.
2. Armstrong, H. G.: Principles and Practice of Aviation Medicine, Baltimore, Williams & Wilkins Company, 1952.
3. Armstrong, H. G., and Heim, J. W.: Effect of Flight on the Middle Ear, *J. A. M. A.* 109:417-421, 1937.
4. Aschan, G. K.: Aero-Otitis Media and Aerosinusitis, *Acta Otolaryng. Supp.* 69:1-93, 1948.
5. Barach, A. L.: The Effect of Low and High Oxygen Tensions on Mental Functioning, *J. Aviation Med.* 12:30-38, 1941.
6. Behnke, A. R.: Physiologic Effect of Pressure Changes with Reference to Otolaryngology, *Tr. Am. Acad. Ophth.* 49:63-71, 1944.
7. Bell, J. J.: Aerotitis Treated by Thermotherapy, *Air Surg. Bull.* 1:10-11, 1944.
8. Bowen, W. J.: Delayed Acute Aero-Otitis Media and Methods of Prevention, *U. S. Nav. Med. Bull.* 44:247-252, 1945.
9. Campbell, P. A.: Aviation Medicine with Reference to the Ear and Upper Respiratory Apparatus (Special Article), *Year Book of Eye, Ear, Nose and Throat*, Chicago, Year Book Publishers, 1941.
10. Campbell, P. A.: Measurement of Eustachian Tube Resistance in Individuals not Subject to Aero-Otitis Media, *USAF School of Aviation Medicine*, Project No. 6 (June) 1942.
11. Campbell, P. A.: Chap. 19 in *Medicine of the Ear*, New York, Thomas Nelson & Son, 1948.
12. Campbell, P. A.: The Importance of the Impedance Formula in the Interpretation of Audiograms, *Tr. Am. Acad. Ophth.* 54:245-252, 1950.
13. Canfield, N., and Bateman, G. H.: Myringo-Puncture for Reduced Intra-tympanic Pressure, *J. Aviation Med.* 15:340-346, 1944.
14. Chang, H. T., Margaria, R., and Gelfan, S.: Pressure Changes and Barotrauma Resulting from Decompression and Recompression in the Middle Ear of Monkeys, *Arch. Otolaryng.* 51:378-399, 1950.
15. Costen, J. B.: A Syndrome of Ear and Sinus Symptoms Dependent upon Disturbed Function of the Temporo-Mandibular Joint, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 43:1-15, 1934.
16. Costen, J. B.: Present Status of the Mandibular Joint Syndrome in Otolaryngology, *Tr. Am. Acad. Ophth.* 56:809-823, 1951.
17. Crowe, S. J.: Irradiation of the Nasopharynx, *Tr. Am. Acad. Ophth.* 51:29-35, 1946.
18. Dickson, E. D. D., et al: Contributions to Aviation Otolaryngology, London, Headley Brothers, 1947.
19. Dickson, E. D. D., and McGibbon, J. E. G.: Otitic Barotrauma, the Treatment of Recurrent, by Irradiation, *J. Laryng. & Otol.* 63:647-671, 1949.
20. Eggston, A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1947.
21. Farrior, J. B.: Histopathologic Considerations in Treatment of the Eustachian Tube, *Arch. Otolaryng.* 37:609-621, 1943.
22. Fowler, E. P., Jr.: Causes of Deafness in Fliers, *Arch. Otolaryng.* 42:21-32, 1945.
23. Fowler, E. P., Jr.: *Medicine of the Ear*, New York, Thomas Nelson & Sons, 1948.

24. Frenzel, H.: Otorhinolaryngology, German Aviation Medicine World War II, Chap. X-A, 977-984, Department of the Air Force.
25. Fulton, et al: Decompression Sickness, Philadelphia, W. B. Saunders Company, 1951.
26. Gidoll, S. H.: New Right-Angle Oscope Facilitating Differential Diagnosis in Tympanic Pathology, A. M. A. Arch. Otolaryng. 54:554-557, 1951.
27. Graves, G. O., and Edwards, L. F.: The Eustachian Tube, Review of Anatomy, Arch. Otolaryng. 39:359-397, 1944.
28. Guggenheim, P.: The Adenoid Problem, A. M. A. Arch. Otolaryng. 55: 146-152, 1952.
29. Haines, H. L., and Harris, J. D.: Aerotitis Media in Submariners, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 55:347-371, 1946.
30. Hall, J. F., Jr.: Use of Helium-Oxygen Mixtures in Aviation for Prevention of Painful Ear Symptoms, J. Aviation Med. 11:81-86, 1940.
31. Hantman, I.: Secretory Otitis Media, Arch. Otolaryng. 38:561-573, 1943.
32. Heller, Mager, and von Schroetter, Luftdruck Erkrankungen, Wein, 1900.
33. Hoople, G. D.: Otitis Media with Effusion, Tr. Am. Acad. Ophth. 54:531-541, 1950.
34. Jones, M. F., and Edmonds, F. C., Jr.: Air Pressure Effects on Hearing and Equilibrium of Unoperated and Fenestrated Ears, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 64:323-344, 1949.
35. Jordan, R. E.: Role of Allergy in Otology, A. M. A. Arch. Otolaryng. 55:363-368, 1952.
36. Kos, C. M.: Effect of Barometric Pressure Changes on Hearing, Tr. Am. Acad. Ophth. 49:75-81, 1944.
37. Lieberman, A. T.: Aero-Otitis Media in Pressure Chamber "Flights," Arch. Otolaryng. 43:500-507, 1946.
38. Lindsay, J. R., and Perlman, H. B.: Tests for Chronic Eustachian Tube Obstruction, Tr. Am. Acad. Ophth. 54:486-491, 1950.
39. Lion, H.: Fold and Crypt Formation in the Nasopharynx, Arch. Otolaryng. 51:655-666, 1950.
40. Loch, W. E.: Effect of Experimentally Altered Air Pressure in Middle Ear on Hearing Acuity in Man, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:995-1006, 1942.
41. Luft, U. C.: Personal Communication.
42. McMyn, J. K.: Anatomy of Salpingo-Pharyngeus Muscle, J. Laryng. & Otol. 55:1-22, 1940.
43. Melvin, W.: Unpublished data, Department of Medical Statistics, USAF School of Aviation Medicine, Randolph Field, Texas.
44. Merica, F. W.: Vertigo Due to Obstruction of the Eustachian Tube, J. A. M. A. 118:1282-1284, 1942.
45. Moffitt, O. P., Lett, J. E., and Tonndorf, J.: Aviation Otolaryngology, USAF School of Aviation Medicine Textbook No. 4, Air University, USAF, 1950.
46. Ogden, F. W.: Aero-Otitis Media in Altitude Chamber Flights, USAF School of Aviation Medicine Research Report No. 147 (May) 1943.
47. Ogden, F. W.: A Study of the Effects of Vasoconstrictor Solutions on Altitude Chamber Aero-Otitis Media, U.S. N.R.C. C.A.M. Report No. 170 (June) 1943.
48. Ogden, F. W.: Modified Politzerization in Treatment of Aero-Otitis Media, USAF School of Aviation Medicine Project No. 173 (Aug.) 1943.

49. Pennington, G. W.: Observations on Chronic Aero-Otitis Media, *Air Surg. Bull.* 2:269-270, 1945.
50. Perlman, H. B.: Eustachian Tube, Abnormal Patency and Normal Physiologic State, *Arch. Otolaryng.* 30:212-238, 1939.
51. Perlman, H. B.: Quantitative Tubal Function, *Arch. Otolaryng.* 38:453-465, 1943.
52. Perlman, H. B.: Mouth of Eustachian Tube: Action During Swallowing and Phonation, *A. M. A. Arch. Otolaryng.* 53:353-369, 1951.
53. Pittman, L. K.: Physiology and Pathology of the Eustachian Tube as Observed by Direct Examination with a Catheterizing Nasopharyngoscope, *Tr. Am. Acad. Ophth.* 50:192-197, 1946.
54. Polvogt, L., and Babb, D. C.: Histologic Studies of the Eustachian Tube of Individuals with Good Hearing, *Laryngoscope* 50:671-675, 1940.
55. Proetz, A. W.: The Displacement Method of Sinus Diagnosis and Treatment, St. Louis, Annals Publishing Company, 1946.
56. Rees-Jones, G. F., and McGibbon, J. E. G.: Radiological Visualization of Eustachian Tube, *Lancet* 2:660-662, 1941.
57. Requarth, W. N.: Aero-Otitis Media in Compressed Air Workers: Treatment with Helium-Oxygen Mixtures, *J. A. M. A.* 116:1766-1769, 1941.
58. Rich, A. R.: Physiological Study of Eustachian Tube and its Muscles, *Johns Hopkins Hosp. Bull.* 31:206-214, 1920.
59. Seaver, E. P., Jr.: The Malocclusion Factor in Obstruction of the Eustachian Tube, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 59:391-398, 1950.
60. Seltzer, A. P.: Newly Designed Paracentesis Needle for Use in Aerotitis Media, *A. M. A. Arch. Otolaryng.* 53:672-674, 1951.
61. Shilling, C. W.: Aero-Otitis Media and Auditory Acuity Loss in Submarine Escape Training, *Tr. Am. Acad. Ophth.* 49:97-102, 1944.
62. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, *U. S. Nav. Med. Bull.* 40:664-685, 1942.
63. Shilling, C. W., Haines, H. L., Harris, J. D., and Kelly, W. J.: Prevention and Treatment of Aerotitis Media, *U. S. Nav. Med. Bull.* 46:1529-1558, 1946.
64. Simkins, C. S.: Functional Anatomy of the Eustachian Tube, *Arch. Otolaryng.* 38:476-484, 1943.
65. Snapp, F. E., and Adler, H. F.: Oxygen Toxicity, USAF School of Aviation Medicine Project Report (Nov.) 1948.
66. Spiegel, F. A.: Aerotitis Treated by Inflation with Helium, *Air Surg. Bull. No. 6*, 1:9-10 (June) 1948.
67. Stewart, C. B., Warwick, O. N., and Bateman, G. L.: Acute Otitic Barotrauma Resulting from Low Pressure Chamber Tests, *J. Aviation Med.* 16:385-408, 1945.
68. Teed, R. W.: Factors Producing Obstruction of the Auditory Tube in Submarine Personnel, *U. S. Nav. Med. Bull.* 42:293-306, 1944.
69. Tonndorf, J.: The Influence of Service on Submarines on the Auditory Organ, *German Submarine Medicine in World War II (D.N.)*, 1947.
70. Trowbridge, B.: A New Treatment of Acute Aero-Otitis Media, *Arch. Otolaryng.* 50:255-263, 1949.
71. Tuttle, A. D.: Passenger Comfort in Commercial Aviation, *J. Aviation Med.* 17:584-589, 1946.
72. Van Dishoeck, H. A. E.: Measurement of the Tension of the Tympanic Membrane and of the Resistance of the Eustachian Tube, *Arch. Otolaryng.* 34:596-602, 1941.

73. Wever, E. G., Bray, C. M., and Lawrence, M. J.: Effects of Pressure in the Middle Ear, *J. Exper. Psychol.* 30:40-52, 1942.
74. Willhelmy, G. E.: Ear Symptoms Incidental to Sudden Altitude Changes, and the Factor of Overclosure of the Mandible, *U. S. Nav. Med. Bull.* 34:533-541, 1936.
75. Wisheart, R. H.: A New Method for Treatment of Acute Aerotitis Media, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 52:581-588, 1943.
76. Wright, R. W.: Aero-Otitis Media, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 54:499-511, 1945.
77. Zoellner, F. v.: *Ohrtrumpete*, Berlin, J. Springer, 1942.
78. Combined Report by the Officers Participating: The Use of Radium in the Aerotitis Control Program of the Army Air Forces, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 54:649-724, 1945.
79. Symposium on Irradiation of Lymphoid Tissue, *Tr. Am. Acad. Ophth.* 54:479-530, 1950.

SUPPLEMENTAL REFERENCES

1. Campbell, P. A.: Effect of Flight Upon Hearing, *J. Aviation Med.* 13:36-61, 1942.
2. Carson, L. D.: Otolaryngologic Aspects of Aviation, *Laryngoscope* 52:706-710, 1942.
3. Dickson, E. D. D., McGibbon, J. E. G., and Campbell, A. C. P.: Acute Otitic Barotrauma, *J. Laryng. & Otol.* 58:465-488; 493-495, 1943.
4. Dickson, E. D. D., McGibbon, J. E. G., Harvey, W., and Turner, W.: Investigation into Incidence of Acute Otitic Barotrauma as Disability Amongst 1000 Aircrew Cadets During Decompression Test, *J. Laryng. & Otol.* 59:267-295, 1944.
5. Hendricks, J. E., and Lieberman, A. T.: Activities of the Irradiation Clinic at Westover Field, Massachusetts, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 54:662-683, 1945.
6. McGibbon, J. E. G.: Aviation Pressure Deafness, *J. Laryng. & Otol.* 57:14-72, 1942.
7. McGibbon, J. E. G.: The Nature of the Valvular Action (Passive Opening) of the Eustachian Tube in Relation to Changes of Atmospheric Pressure and to Aviation Pressure Deafness, *J. Laryng. & Otol.* 57:344-350, 1942.
8. Ogden, F. W.: Continuous Pressure Politzerization in the Prevention of Altitude Chamber Aero-Otitis Media, *U.S. N.R.C. C.A.M. Report No. 170* (June) 1943.
9. Ogden, F. W.: Politzerization, A Simple and Effective Method of Treatment of Aero-Otitis Media, *Air Surg. Bull.* 1:18-20, 1944.
10. Perlman, H. B.: Observations on the Eustachian Tube, *A. M. A. Arch. Otolaryng.* 53:370-385, 1951.
11. Shilling, C. W., Haines, H. L., Harris, J. D., and Kelly, W. J.: Aerotitis Media; Brief Presentation of its Symptomatology, Prevention and Treatment, *J. Aviation Med.* 18:48-55, 1947.
12. Simpson, J. F.: General Survey of Otorhinolaryngological Considerations in Service Aviation, *J. Laryng. & Otol.* 57:1-7, 1942.
13. Whaley, J. B.: Problems of Aviation Medicine Relating to Ear, Nose and Throat, *Arch. Otolaryng.* 36:438-442, 1942.

LXXVII

PROBLEMS IN DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE LOWER PORTION OF THE ESOPHAGUS AND THE CARDIA

HERMAN J. MOERSCH, M.D.

ROCHESTER, MINN.

Many difficulties may be encountered in the field of endoscopic diagnosis. It is a topic with which your essayist regrets to say he has had considerable experience. The difficulties are of such magnitude and ramifications that it would be impossible to discuss all of them at this time. I shall limit my remarks, therefore, to some of the problems involved in the differential diagnosis of lesions of the lower end of the esophagus and the cardia.

The lower portion of the esophagus and the cardia fall heir to a great variety of organic, developmental and psychosomatic disturbances which often present extremely difficult problems in differential diagnosis.

CARDIOSPASM

In most cases cardiospasm can be recognized from the clinical history and roentgenologic examination alone, but occasionally it may be closely mimicked by other types of esophageal disease. Cardiospasm also may assume a variety of forms which increases the difficulty of diagnosis. It may vary from slight obstruction at the cardia with minimal dilatation of the esophagus (Fig. 1*a*) to marked obstruction at the cardia with enormous dilatation and angulation of the esophagus above (Fig. 1*b*). When a sound is passed over a previously swallowed silk thread, an obstruction of a pliable nature may be encountered at the cardia which can readily be overcome by gentle pressure. Should the obstruction be firm or hard, the possibility that it is caused by a neoplasm should be seriously considered.

In case of doubt as to the diagnosis, esophagoscopy should always be performed. The esophagoscopic findings will vary with the degree and duration of esophageal obstruction. If extreme angulation occurs in the lower end of the esophagus, some difficulty may be experienced in passing the esophagoscope through the cardia and into the stomach

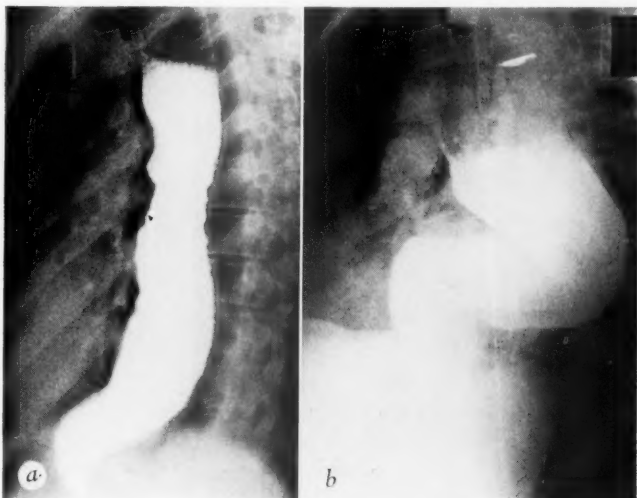


Fig. 1.—*a*. Cardiospasm with minimal dilatation of the esophagus.
b. Cardiospasm with extreme dilatation and angulation of esophagus.

and the correctness of the diagnosis may be doubted. In cases of this type, and especially in the early stages of cardiospasm when the findings are minimal, intramuscular administration of methacholine chloride (mecholyl chloride) may be a great diagnostic aid. Kramer and Ingelfinger¹ found that if a patient with cardiospasm is given an intramuscular injection of mecholyl a tetanic contraction of the esophagus occurs and the patient suffers from severe substernal distress. This reaction occurs if cardiospasm is present, regardless of the degree of esophageal dilatation, but does not occur in any other type of esophageal disease. My experience with mecholyl in the treatment of esophageal disease has been identical with this observation in most essential aspects.

DIFFUSE SPASM

Diffuse spasm of the lower third of the esophagus is frequently confused with cardiospasm. It may give rise to symptoms that closely mimic those produced by cardiospasm. It is important that these two conditions be distinguished from each other because of the difference in treatment.

Roentgenologic examination of the esophagus reveals evidence of diffuse spasm involving the lower third of the esophagus, usually



Fig. 2.—Diffuse spasm of the lower third of the esophagus.

associated with slight dilatation of the upper two thirds (Fig. 2). The esophagoscopy findings vary according to whether the examination is performed with the patient under topical or general anesthesia. If it is carried out with the use of general anesthesia, nothing other than a mild degree of esophagitis may be encountered. With the patient under topical anesthesia, the lower third of the esophagus will be found closed. The obstruction can be overcome gradually by applying gentle pressure to the instrument as it is advanced. On passage of a sound over a previously swallowed silk thread, an obstruction will be encountered in the lower portion of the esophagus. Gentle pressure on the sound will cause the obstruction to give way and the sound will advance slowly through the esophagus but on its way into the stomach it will be grasped repeatedly by the esophagus. If the dilatation is carried out with the patient under general anesthesia the sound can usually be passed without encountering any obstruction.

The following report of a case illustrates how closely diffuse spasm of the lower third of the esophagus can simulate cardiospasm, and the effect of general anesthesia on diffuse spasm:



Fig. 3.—Diffuse spasm of the lower portion of the esophagus simulating cardiospasm.

Fig. 4.—Carcinoma of cardia simulating cardiospasm.

CASE 1.—A woman, 63 years of age, had been well until one year previous to her registration at the Mayo Clinic. At this time watery material regurgitated into her mouth when she was lying down and food would lodge at the level of the suprasternal notch. Solid food caused more difficulty than liquids. Roentgenologic examination of the esophagus elsewhere was reported to show evidence of an obstruction in the lower part of the esophagus. Esophagoscopy also had been carried out with the patient under topical anesthesia and was reported to reveal an obstruction of the lower end of the esophagus which could not be overcome by pressure on the esophagoscope or passage of bougies.

Three days after coming to the clinic a roentgenogram of the esophagus demonstrated an obstruction of the lower portion with some dilatation of the upper portion (Fig. 3). Esophagoscopy, which was done with the patient under anesthesia produced by intravenous administration of pentothal sodium, failed to show any evidence of obstruction and the esophagoscope could be passed into the stomach without difficulty. A day later, a sound passed over a previously swallowed silk thread without the use of anesthesia revealed a spasmotic obstruction involving the lower end of the esophagus.

CARCINOMA

Carcinoma of the lower end of the esophagus and cardia may offer a great deal of difficulty in diagnosis. This is especially true in those cases in which carcinoma arises in the cardia and infiltrates under the esophageal mucosa, producing esophageal obstruction without evidence of ulceration or tumefaction. Although, as a rule, carcinoma does not give rise to marked dilatation of the esophagus proximal to the site of obstruction, at times the dilatation may be of such magnitude that it will closely simulate that associated with cardiospasm. Every effort should be exercised to obtain a satisfactory specimen for biopsy. Experience has demonstrated, however, that in approximately 10 per cent of carcinomas of the esophagus and 25 per cent of carcinomas of the cardia, biopsies will be noninformative. Considerable help may be obtained in dealing with this problem by removing secretions or smears from the site of obstruction for cytologic examination. It is possible, by combining cytologic studies with biopsies, not only to make diagnosis in a high percentage of carcinomas, but also, in my opinion, to recognize the lesion at an earlier stage than it can be recognized by biopsy alone.

The importance of microscopic examination of tissue removed from a carcinoma in arriving at a correct diagnosis is well exemplified by the following case:

CASE 2.—A man, 31 years of age, had been well until eight months before coming to the clinic, when he first noted that food would lodge in the lower portion of his esophagus and that he would have to regurgitate the food to get relief. These symptoms began the same day that he had received unpleasant news, and it was assumed that his difficulty was functional in origin. This diagnosis seemed to be confirmed when roentgenologic examination of the esophagus failed to show any evidence of trouble. At the onset, liquids seemed to cause obstruction much more frequently than solids. Later, however, he had had more and more difficulty with solid food until at the time of his admission to the clinic he was unable to swallow anything but liquids. He had lost 80 pounds (36.3 kg).

Roentgenologic examination of the esophagus showed evidence of cardiospasm (Fig. 4). On passage of a sound over a previously swallowed silk thread, a firm obstruction was encountered at the cardia. Esophagoscopy revealed a polypoid lesion obstructing the cardia. Microscopic examination of a specimen removed from the lesion showed the obstruction to be an adenocarcinoma, grade 4.

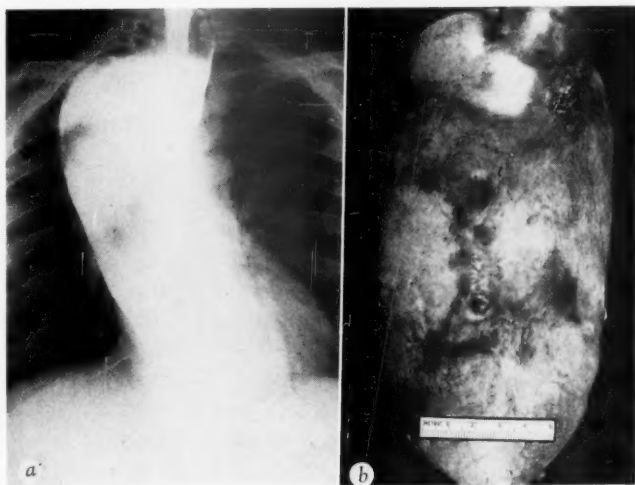


Fig. 5.—*a*. Lipoma of the esophagus presenting roentgenographic evidence similar to cardiospasm. *b*. Resected specimen of lipoma.

My colleagues and I have found that in cases of small carcinomatous lesions of the cardia valuable information often may be obtained by passage of sounds. Not only is it possible to detect such lesions on palpating the cardia with a sound, but occasionally smears taken from the sound may afford cytologic evidence of a carcinoma.

This case illustrates well that roentgenologic examination alone is not reliable for the diagnosis of carcinoma of the cardia. It also emphasizes how closely carcinoma can simulate cardiospasm and the necessity of complete examination of any patient who has a history of dysphagia.

BENIGN TUMORS

Although benign tumors of the esophagus are not common, when they do occur, occasionally they offer great difficulty in differential diagnosis. This is true not only of mucosal tumors but also of intramural, extramucosal tumors. The following case is an example of a mucosal tumor which was confused with cardiospasm:

CASE 3.—A man, 42 years of age, was referred to the clinic for treatment of cardiospasm. He had been well until eight years previously, when he had first noted regurgitation of food after eating a large meal. During the next two or three years he had had

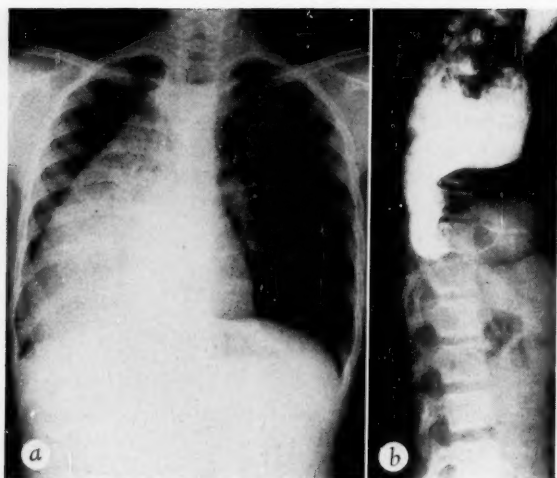


Fig. 6.—*a*. Leiomyoma obstructing lower end of esophagus with marked dilatation of the esophagus. *b*. Note the peculiar contour of the wall of the lower portion of the esophagus in leiomyoma of esophagus.

moderate dysphagia. About a month after the onset of dysphagia he had felt a mass come up in the back of his throat after he had vomited. He had tried to swallow the mass, which he thought was about the size of a large lemon, but as he had been unsuccessful in doing this he had pushed it down with his fingers. After this, he had begun to have more trouble with gaseous eructations, sore throat and regurgitation of undigested food. A diagnosis of cardiospasm had been made by his home physician. An esophagoscope had been used and the esophagus had been dilated with a mercury dilator a number of times without benefit. During the year preceding his admission to the clinic, the patient had had great difficulty with regurgitation. This was especially severe when he attempted to lie down to sleep, and it was necessary for him to sleep in the upright position.

A roentgenogram of the esophagus at the clinic was reported as showing evidence of cardiospasm (Fig. 5*a*). Esophagoscopy examination, however, revealed a large, pedunculated tumor covered with normal mucosa. The tumor was attached by a pedicle to the upper end of the esophagus, immediately below the cricoid cartilage, and extended down to the cardia. The tumor was removed sur-

gically through a right transpleural approach and proved to be a large lipoma (Fig. 5*b*).

Intramural, extramucosal esophageal tumors likewise may present a clinical history and roentgenologic appearance suggestive of cardiospasm, as illustrated by the following case:

CASE 4.—A girl, 13 years of age, had been well until six years prior to her admission to the clinic. At that time she had begun to vomit her food soon after completion of a meal. At first this had occurred only intermittently, but during the year previous to coming to the clinic, it had occurred after all meals except breakfast. Solid foods seemed to cause more difficulty than liquids. In spite of her difficulty the patient had maintained an excellent appetite and had lost little weight.

Roentgenologic examination of the esophagus showed evidence of a tremendously dilated esophagus with obstruction at the esophogogastric junction (Fig. 6*a* and *b*). The radiologist was unable to distinguish between cardiospasm and an organic lesion at the cardia with secondary dilatation of the esophagus. The peculiar contour of the wall of the lower portion of the esophagus strongly suggested that the difficulty might be due to a leiomyoma involving that portion. On esophagoscopy examination, the esophagus was found to be distended with more than 1,000 cc of liquid mixed with food. After removal of this material the esophageal mucosa showed marked evidence of esophagitis. Although the possibility of a leiomyoma involving the lower end of the esophagus was considered, no such lesion could be demonstrated. An attempt was made to relieve the esophageal obstruction by dilatation with sounds, but this proved unsuccessful. The patient was operated on and was found to have a large multicentric leiomyoma involving the lower portion of the esophagus. The tumor completely encircled the lower end of the esophagus and obstructed the esophageal lumen at the esophageal hiatus.

HIATAL HERNIA

Hiatal hernia frequently causes difficulty in differential diagnosis of lesions involving the cardia. This difficulty is accentuated by the fact that hiatal hernia not only may vary in size and type but also owing to ulceration, which is such a frequent accompaniment of this condition, may stimulate secondary spasm of the esophagus. The problem is complicated further by the fact that the hiatal hernia may not be the primary lesion but a secondary manifestation of such diseases as carcinoma, scleroderma, esophagitis and cicatricial



Fig. 7.—Hiatal hernia with carcinoma at esophagogastric junction.

stricture of the esophagus. Finally, foreign bodies may become lodged in a hiatal hernia, or other diseases may develop in a hiatal hernia and greatly alter the findings. The following two cases are examples of such changes.

CASE 5.—A woman, 50 years of age, had been well until 10 months previous to her registration at the clinic when she had first noticed pain under the lower end of the sternum on swallowing. At that time the pain was not severe and lasted only a few moments, but gradually it had increased in intensity and duration, and by the time she presented herself for examination it would last for two hours. She had never had any dysphagia. The home physician had passed rubber tubes through the esophagus at various times but without benefit to the patient. A diagnosis of cardiospasm had been made.

Roentgenologic examination of the esophagus was reported to show evidence of a hiatal hernia, with the upper half of the stomach above the diaphragm, and some narrowing of the esophagogastric junction (Fig. 7). Esophagoscopy revealed a large hiatal hernia. Immediately below the esophagogastric junction an ulcerating lesion was found. A section of tissue was removed from the lesion and, on microscopic examination, it was reported to show evidence of inflammation. Because of the suspicious appearance of

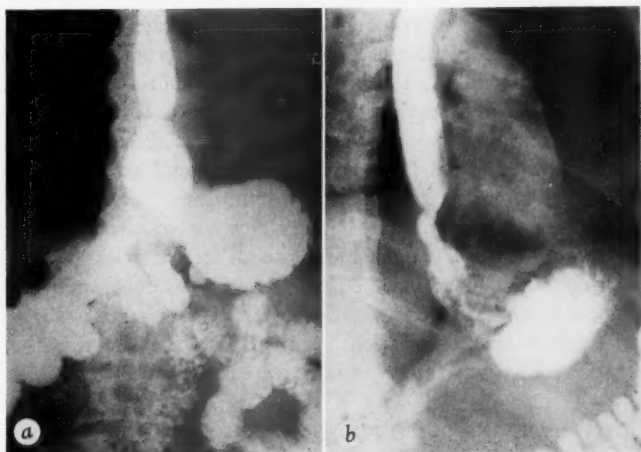


Fig. 8.—*a*. Hiatal hernia with foreign body lodged at esophagogastric junction. *b*. Same case after removal of persimmon.

the ulcerating lesion, esophagoscopy was repeated and further tissue was removed for microscopic examination. A diagnosis was made of squamous cell carcinoma, grade 3. Operation was performed and the patient was found to have an esophageal hiatal hernia associated with carcinoma which involved the esophagogastric junction.

In the second case the results of esophagoscopy proved to be far more satisfactory.

CASE 6.—A man, 63 years of age, had been well until 18 months before coming to the clinic. At that time he suddenly had had trouble with solid food lodging in the region under the lower end of his sternum. If he chewed his food thoroughly he had little difficulty, but he did notice that the size of the particles of food he could swallow comfortably became smaller and smaller. A year prior to his admission roentgenograms had been made of the esophagus and were reported to show evidence of carcinoma involving the lower end of the esophagus. Esophagoscopy had been performed and carcinoma was reported to involve the lower portion of the esophagus although no specimen had been removed for biopsy. The patient had been given a course of roentgen therapy over the site of the lesion. Since then, he had noted gradually increasing difficulty with swallowing and regurgitation. During the year prior to his admission he had lost 40 pounds (18.1 kg) .

A roentgenogram of the esophagus at the time of the patient's admission to the clinic was reported as showing evidence of carcinoma of the lower third of the esophagus with obstruction (Fig. 8a). Esophagoscopy examination revealed a large necrotic-appearing mass obstructing the lower portion of the esophagus. When an attempt was made to remove tissue from the mass for microscopic diagnosis, it was noted that the material obtained appeared to contain fiber. After some manipulation the entire tumor mass was removed and was found to be a persimmon! After removal of the foreign body it was found that the patient had a hiatal hernia with stricture at the esophagogastric junction. This diagnosis was confirmed on subsequent roentgenologic examination of the esophagus (Fig. 8b). It is of interest that the patient recalled that the last time he had eaten persimmons was the day his dysphagia had begun.

OTHER LESIONS COMPLICATING DIFFERENTIAL DIAGNOSIS

Congenital anomalies of the lower portion of the esophagus, esophageal varices, esophagitis, tuberculosis, fungous diseases, and post-traumatic changes of the esophagus may give rise to clinical symptoms similar to those of cardiospasm. They may offer difficulty in distinguishing them not only from cardiospasm but also from each other.

It is obvious that difficulties often may be experienced in the differential diagnosis of lesions involving the lower portion of the esophagus. Some of these difficulties have been pointed out, as well as the important role which the esophagologist must play in dealing with these problems and some of the tools he may use in differential diagnosis.

MAYO CLINIC.

REFERENCE

1. Kramer, Philip, and Ingelfinger, F. J.: Cardiospasm, A Generalized Disorder of Esophageal Motility, *Am. J. Med.* 7:174-179 (Aug.) 1949.

LXXVIII

THE PATHOLOGY, SYMPTOMATOLOGY AND DIAGNOSIS
OF CERTAIN COMMON DISORDERS OF
THE VESTIBULAR SYSTEM

M. R. DIX, F.R.C.S.

AND

C. S. HALLPIKE, F.R.C.P., F.R.C.S.

LONDON, ENGLAND

It is generally agreed that within the last fifteen years some extension has been achieved of our understanding of the problems of human vertigo. Nevertheless, difficulties and confusion still persist and in the course of the present communication an attempt will be made to advance the process of its clarification, both by critical review and by a short account of some of our recent investigations. The foundations of the subject are to be found in the writings of Prosper Ménière, and these we take as our starting point.

Ménière's papers on vertigo are chiefly remarkable for the powers which their writer displays of describing and analysing the symptoms and signs of disease. It is easy now to realise that it was this mastery of symptomatology which, more than anything else, enabled him to identify, with an accuracy which has never been seriously questioned, the disease of the labyrinth which has since come to bear his name. Beyond, however, asserting with confidence that the disease was due to a lesion of the internal ear, limited to that organ and indestructive to life, he made no direct observations upon its pathological anatomy, and further information upon this point was not forthcoming until 1938. Since then the morbid anatomical basis of the disease has been established by means of histological examinations of the temporal bones in a number of clinically characteristic cases. It is fair to say that this opinion is not perhaps a universal one. On the contrary it is still sometimes said that Ménière did describe the pathological anatomy of Ménière's disease, and reference is made to the case which

From the Aural Department and the Otological Research Unit of the Medical Research Council, National Hospital, Queen Square, London.

Read before the Section of Otology, Royal Society of Medicine, March, 1952.

Ménière cited of the young girl who died after a short illness, due to a chill, in the course of which she suffered from deafness, vertigo and vomiting. Ménière examined the temporal bones and found a reddish exudate in the semicircular canals. But no reader of his text could suppose that Ménière wished it to be inferred from this observation that the cause of *this* girl's illness was the same as that responsible for the recurrent attacks of vertigo with deafness so characteristic of the other, but essentially benign, disease which he had described so clearly. Indeed, the fatal issue of the case alone rules out this possibility.

Our own view is that Ménière's reference to the anatomical changes in these labyrinths had an entirely different significance and one, moreover, which is quite obvious from the context.

It is necessary to bear in mind that in 1861, when Ménière published his best known paper, the very possibility that a lesion of the internal ear could cause such severe symptoms as vertigo and vomiting was still a moot point.

Flourens' experimental work on pigeons, in which he demonstrated for the first time that gross disorders of the equilibrium could be produced by injury of the semi-circular canals, had only been published some 30 years earlier, and its significance as regards the problems of human disease had not yet been appreciated. It was Ménière's great merit that he knew of Flourens' work and understood its meaning. To him, therefore, the significance of the exudate in the semi-circular canals of his patient was that in it he recognised the vital link so far missing between Flourens' animal experiments and the problem of human vertigo, a demonstrable lesion of the semi-circular canals in a subject who had suffered in life from vertigo. Ménière argued that if such a hemorrhagic lesion, occurring in the course of this girl's fatal illness, could produce vertigo, then other lesions of the inner ear, be their precise nature what you will, could also be the cause of the vertigo in his other group of subjects whose symptoms he had described with such care. There Ménière left the matter. He had identified his disease by his accurate specification of its symptomatology and natural history. As to its pathology he said only this: 'La lésion matérielle réside dans les canaux semi-circulaires' (the essential lesion is situated on the semi-circular canals.) Little more knowledge came our way until 1938 when Cairns and Hallpike examined the temporal bones of their two subjects and established the nature of Ménière's lesion.¹

Of this the essential abnormality was a distension without evidence of infection or trauma of the endolymphatic vesicle; a finding,

remarkable in itself and since reproduced with remarkable uniformity in a considerable number of further histological studies of the temporal bones carried out by Hallpike and also by a number of others.

To summarize this chapter of history it can be said that Ménière's concept was of a disease, *sui generis*, of the internal ear. This he based in the main upon his own studies of symptomatology and supported it by certain indirect evidence of animal experimentation and of human pathology. Since 1938 this concept has been abundantly confirmed by further clinical studies and by direct pathological evidence derived from the study of human temporal bones.

This analysis of Ménière's work and the position it occupies in the much larger field of organic vertigo as we know it today was clearly stated by Cairns and Hallpike in their original paper, and has since gained wide acceptance by a number of American authors, in particular Day,² Wells³ and Williams.⁴ But since Ménière's time this field has become much enlarged and its contours very confused and it has been customary to mix together with Ménière's disease, under the indiscriminating label of Ménière's Syndrome or even of pseudo-Ménière's disease, other types of organic vertigo which conform only vaguely to the established symptomatology and pathology of Ménière's disease. In the past this has been unavoidable, and connotes only our ignorance of the clinical and pathological features of these disorders which would enable us to distinguish them from each other and from Ménière's disease.

It is, of course, the task of oto-neurological research to resolve this ignorance. When complete, it will be possible so to distinguish them and call them by names which will announce their clinical and pathological individualities. When that time comes the need for the label, Ménière's Syndrome, will disappear and no more will be heard of that lexicographical abomination, 'pseudo-Ménière's disease.' The task has been begun with the clinico-pathological work carried out during the last fifteen years upon Ménière's disease. During this, we have seen rescued from the confusion what Ménière put into it, his disease, with its symptomatology confirmed and its pathology established. It is the purpose of the present communication to present certain new information upon the subject of Ménière's disease. In addition, descriptions will be given of two other varieties of organic vertigo; one a disorder of the vestibular neurones; another a disorder of the otolith system in the labyrinth. Both of these are liable to be confused with Ménière's disease, and carry Ménière's name on that familiar label 'Ménière's Syndrome.' It will, however, be shown

that their clinical and pathological features make obvious their distinction from Ménière's disease, and other labels will accordingly be proposed for them which declare this distinction and are more appropriate to their symptoms, signs and pathological anatomy.

In the present work we have endeavored first and foremost to make use of the clinical methods in which Ménière himself so excelled. That is to say, it has been our aim to base our opinions primarily upon the study of symptoms and natural history of disease. To this we have added as much evidence as possible from the physical signs which can now be derived from modern tests of cochlear and vestibular function. Finally, when the opportunity has presented itself we have been able to check our clinical evidence by histological studies of the temporal bones.

MÉNIÈRE'S DISEASE

On symptomatology there is little to add to Ménière's original description and to the analysis published in 'Brain' in 1942 by Cawthorne, Fitzgerald and Hallpike.⁵ We make more, perhaps, of distortion of hearing than Ménière did, and the exacerbation of tinnitus and deafness during the attacks. But these can hardly be described as major developments. As regards physical signs we find the vestibular test results are still very much as previously stated.⁵ In particular the caloric test results are abnormal in 94% of cases. Of these, 20% show a directional preponderance towards the sound ear. In 58% there is a loss of canal sensitivity in the affected ear. In 16% the result represents a mixture of these two primary derangements. In the field of tests of cochlear function, however, we are able to report considerable progress since 1942. Dix, Hallpike and Hood⁶ showed in 1948 that the phenomenon of loudness recruitment was invariably present in Ménière's disease, and that in a proportion of such subjects over-recruitment made its appearance. In another series of observations Hood⁷ has been able to show that the phenomenon of adaptation is abnormally rapid in cases of Ménière's disease. Speech audiometry, too, has in many cases given results in cases of Ménière's disease which have a high diagnostic significance. In particular they reveal a loss of intelligibility which is out of proportion to the pure tone audiometric threshold loss. It will be recalled that in the publication to which reference is made,⁶ the characteristically positive finding of recruitment in Ménière's disease was compared with its characteristic absence in cases of VIIIth nerve tumour, and thereon was based our opinion that the phenomenon of recruitment was attributable to hair cell disease. For this argument

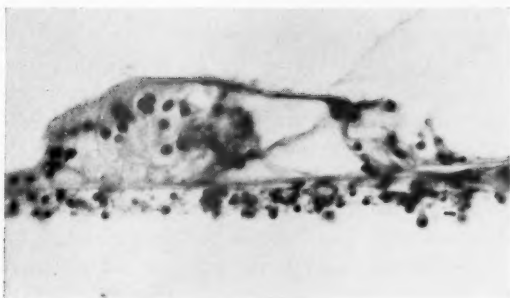


Fig. 1.—Organ of Corti, Normal Cochlea. The organ is of normal form and size, with good preservation of Corti's tunnel. Details of the hair cells are obscured by postmortem degeneration.

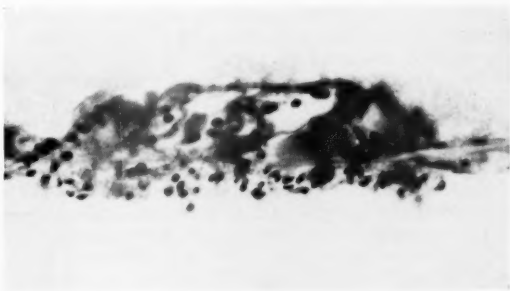


Fig 2.—Organ of Corti. Affected cochlea. The organ is shrunken and its outline irregular. Corti's tunnel is occupied by a structureless coagulum.

pathological evidence is in some cases very strong. That is to say, the cells of Corti's organ show very striking changes while the cochlear nerve fibers and the cells of the spiral ganglion are quite normal. This is exemplified in the accompanying photomicrographs of the organ of Corti in the normal and unaffected ears in the first of the two cases of Ménière's disease described (Figs. 1 and 2).

In other cases, however, examined both by ourselves and others, these changes in the hair cells are by no means obvious, and to circumvent the difficulty it has been necessary to have recourse to the argument that in such cases the histological changes responsible for the deafness and recruitment were of a kind which made it possible for them to be masked by the considerable histologic artefacts which are so often unavoidable with human material. If this argument be accepted, then with it must also be accepted the likelihood that in a case of unilateral Ménière's disease, in which this histological artefact was reduced by exceptionally favorable conditions of fixation, it should be possible to distinguish some significant anatomical changes in the hair cells. A brief account of clinical and pathological data derived from such a case now follows:

The case was that of a man of 54 who died in 1949. In 1938 he began to suffer from attacks of paroxysmal vertigo, during which objects rotated vertically. They were accompanied by nausea and lasted from 10 to 30 minutes. Consciousness was not lost during the attacks during which he was compelled to lie down. He had experienced such attacks yearly until 1945. During that time he also had buzzing tinnitus in the right ear and deafness which was progressive. He was seen at Sir Charles Symonds' Out Patients in November 1948 on account of epileptic attacks lasting for half-an-hour or more, during which he became unconscious. Soon after, he developed drowsiness, confusion and a severe affection of speech. He was admitted to Queen Square on February 9, 1949, where a diagnosis was made of a left temporal lobe neoplasm. This was confirmed by biopsy; the patient died 6 days later. At post-mortem a large tumor was found in the left temporal lobe which histological examination showed to be an astrocytoma. Apart from some herniation of the inner edge of the left temporal lobe through the incisura tentorii on the left hand side and some cerebellar coning, there was no other abnormality of the cerebrum, cerebellum, brain stem, cranial nerves or meninges. The last otological examination was carried out at Guy's in December 1948 when the findings confirmed the diagnosis of Ménière's disease, the nose and throat being found healthy, the tympanic membranes normal with a severe degree of deafness of the right ear of the perceptive type. Pure tone audiometry showed an approximately uniform hearing loss on the right side of some 60 to 70 decibels. There was, in addition, a slight high-tone loss in the left ear confined to the frequencies 4,000 and 8,000 cycles. Caloric responses showed a slight reduction of the responses to both cold and hot stimuli on the right hand side. We were able to obtain the temporal bones 16 hours after death, and to undertake their fixation and preparation ourselves. It was possible to display the histology of the labyrinths in a comparatively good state of preservation; in particular, the condition of the hair cells was sufficiently free from post-mortem artefact to make worthwhile a close morphological comparison of Corti's organ in the two ears.

In Fig. 3 is shown the unaffected cochlea with a normal spiral ganglion and a well preserved organ of Corti with Reissner's mem-

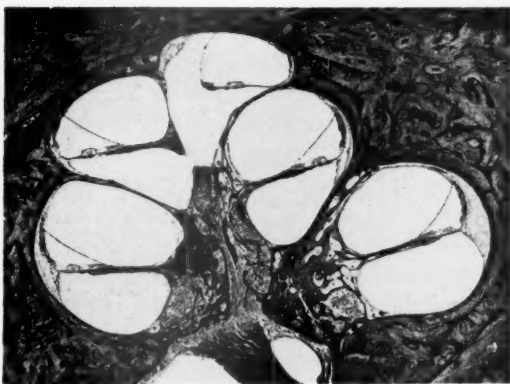


Fig. 3

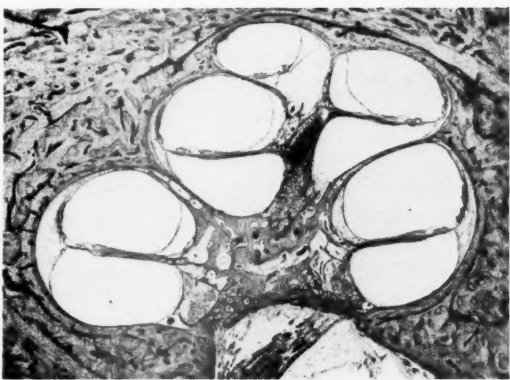


Fig. 4

brane in its normal position. In Fig. 4 is shown the affected cochlea; the spiral ganglion is of normal density. The general structure of Corti's organ can be made out, although not in any great detail. Finally, the typical distention of the scala media is seen with the displacement of Reissner's membrane.

To facilitate comparison of the structure of Corti's organ in these two labyrinths, photomicrographs have been prepared in which views of the organ in the different coils of the unaffected cochlea are presented at high magnification side by side with their counterparts in the affected cochlea.

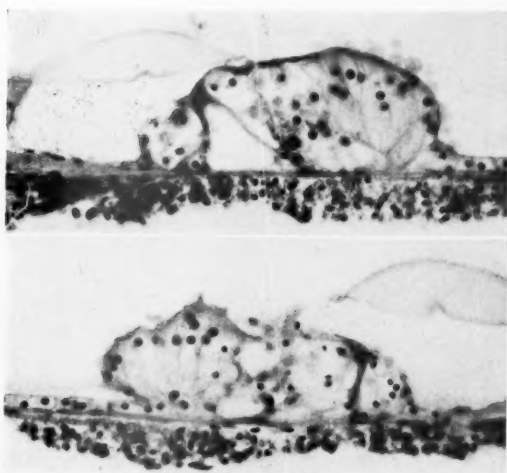
In Fig. 5 are shown the organs of Corti in the anterior and posterior middle coils. On the normal side, the normal form and size of Corti's organ is well preserved in both coils. Both rods of Corti are well seen, and the form of Corti's tunnel is nearly normal. In both coils can be seen what is probably the remains of a hair cell and its nucleus. On the affected side obvious changes are present. Thus, the total size of Corti's organ is reduced, its shape is irregular, the outer rod has been demolished and the hair cell framework has disappeared. In addition, Corti's tunnel, or what is left of it, seems to be occupied by a kind of coagulum.

In Fig. 6 is shown the anterior and posterior basal coils. On the normal side, Corti's tunnel is preserved and the organ is of normal size and shape. There is distinct evidence of at least one hair cell nucleus, while a nerve filament is to be seen crossing Corti's tunnel. On the affected side, however, the outer rod of Corti has disappeared and some coagulum is present in the space of what was Corti's tunnel. Finally, the area occupied by the hair cells seems to be disorganised, both as regards its shape and its cellular contents.

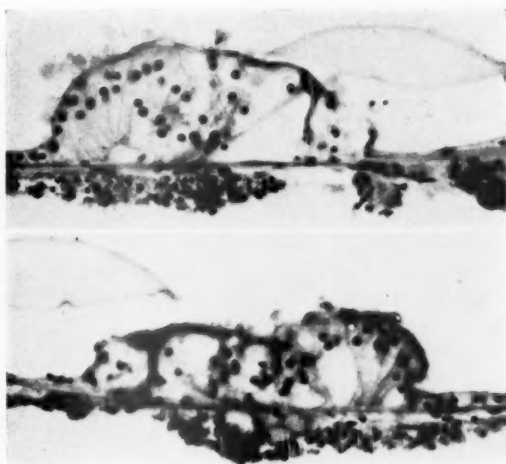
These changes have been demonstrated in some detail since they strengthen in a very satisfactory manner, what has been a weak link in the chain of argument, which has led us to conclude that the deafness in Ménière's disease and the loudness recruitment phenomenon which is so characteristic of it are attributable to hair cell disease.

VESTIBULAR NEURONITIS

We come next to another group of patients whose chief symptom is again vertigo, usually but not always paroxysmal in character. This group is chiefly distinguished from Ménière's disease on clinical grounds by the conspicuous absence of cochlear signs and symptoms. We began to recognize this condition at our Clinic at Queen Square as a distinct clinical entity in about 1946. For a variety of reasons, to which further reference will be made, it seemed then attributable,

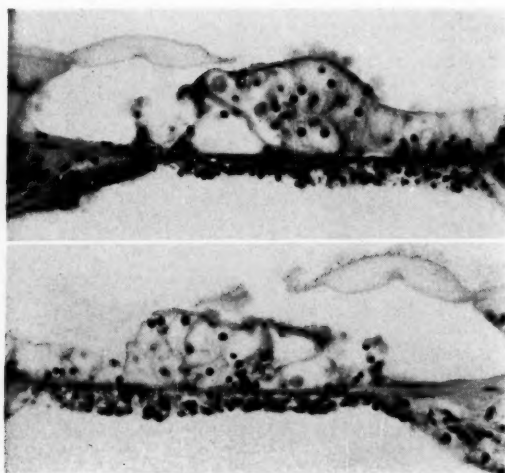


Anterior Middle Coil.
(Upper) Normal Cochlea.
(Lower) Affected Cochlea.



Posterior Middle Coil.
(Upper) Normal Cochlea.
(Lower) Affected Cochlea.

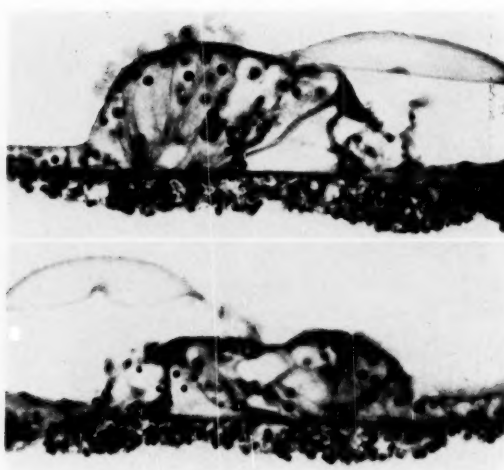
Fig. 5.



Anterior Basal Coil.

(Upper) Normal Cochlea.

(Lower) Affected Cochlea.



Posterior Basal Coil.

(Upper) Normal Cochlea.

(Lower) Affected Cochlea.

Fig. 6.

beyond any doubt, to some form of organic disease confined to the vestibular apparatus and localized, in all probability, to its peripheral nervous pathways up to and including the vestibular nuclei in the brain stem. It was impossible, however, to go further and specify the particular elements of the neurones, cells or fibres, which were affected. When, therefore, it came to naming the condition we required a term comprehensive enough to encompass this uncertainty. We chose the name 'vestibular neuronitis' and have since continued to use it.

We first described the condition in 1949 in a short communication to the Fourth International Congress of Oto-laryngology in London⁸ and our present analysis of its clinical features is based upon the study, made possible through the courtesy of our colleagues at Queen Square, of over 100 cases. These we have examined in the course of the last few years. Age and sex distribution are tabulated as follows:

TABLE I.

Age (Years)	AGE DISTRIBUTION						Total
	Under 20	20-39	30-39	40-49	50-59	Over 60	
Number	5	21	37	22	14	1	100

SEX DISTRIBUTION

Male	57	Total	100
Female	43		

It will be seen that the disorder chiefly affects the age group 30 to 50 without preference for sex. Apart from the absence of cochlear signs and symptoms the condition is often but not always distinguishable from Ménière's disease by the character of the vertigo. This may consist of sudden and transient seizures accompanied by sensations of blackout. On the other hand there may be no severe paroxysms and the disequilibrium may take the form of "feeling top heavy" or "Off-balance," particularly when walking or standing. As in other forms of organic vestibular disease, the disequilibrium is aggravated by head movements of all kinds. In a fairly high proportion of the subjects the onset of the symptoms is associated with some kind of febrile illness, or with evidence of infection of the ears, nose and throat, and to this aspect of the malady we shall later return.

Otoscopic findings are typically normal with normal test results of cochlear function including pure tone audiometry.

When we come to investigate vestibular function, however, very marked abnormalities are always present, in particular of the caloric responses which are consistently reduced, often grossly so, and on both sides. A full description of the caloric abnormalities follows: All tests were carried out in accordance with the technique described by Fitzgerald and Hallpike.⁹ We have divided out test results in accordance with the following nomenclature:

(1) *Complete canal paresis.* No response obtained with irrigation at 20° C. for one minute.

(2) *Severe canal paresis.* No response obtained with stimuli of normal strength, i.e. 30° C. or 44° C. for 40 seconds; a response, however, was obtained with water at 20° C. for one minute.

(3) *Moderate canal paresis.* In this group responses, although obviously reduced, were obtained with stimuli of normal strength.

Certain of the cases exhibited directional preponderance, and others a combination of directional preponderance and canal paresis. For further particulars of the qualitative and quantitative assessment of abnormalities of the patterns of the caloric test results reference should be made to previous publications.^{5, 10}

The abnormalities were further divided into two main groups:

1. Bilateral. 2. Unilateral.

The complete classification is given in Table II.

It will be seen from Table II that substantial abnormalities of the caloric responses were present in all of our 100 cases. In 47 they were bilateral; in 53 they were unilateral.

In a few of the cases the abnormalities of the caloric test findings have been shown by follow-up to be the first and only manifestation of disseminated sclerosis. In the great majority, however, there was no evidence whatsoever of extra vestibular nervous disease, and in these we have postulated an organic lesion of the vestibular nervous pathways at some point up to and including the vestibular nuclei, a vestibular neuronitis. We have preferred to believe that the lesion is central to the labyrinth in accordance with a well established principle of oto-neurology, namely that destructive labyrinthine lesions,

TABLE II.—CALORIC ABNORMALITIES.

BILATERAL	
Complete canal paresis	7
Severe canal paresis	25
Moderate canal paresis	7
Combined canal paresis with directional preponderance to the same side	8
Total	47
UNILATERAL	
Complete canal paresis	8
Severe canal paresis	9
Moderate canal paresis	20
Combined canal paresis with directional preponderance to the opposite side	8
Directional preponderance to the opposite side	8
Total	53

whatever their pathology tend on the whole to involve the cochlear apparatus. Furthermore, a high proportion of our subjects have been found to exhibit significant changes of the galvanic test responses, evidence which is strongly indicative of a lesion central to Scarpa's ganglion. Later reference will be made to the technique and interpretation of these tests.

The condition is essentially a benign one. It responds well to treatment of focal infection when this is present, and generally recovers in the course of a few years. In a few cases we have observed the re-establishment of the caloric responses.

We propose now to consider in greater detail two important clinical aspects of the condition: (1) The role of infection as a pathogenic factor; (2) the galvanic test results and their localising value.

(1) *The role of infection as a pathogenic factor.* The role of focal infection as a cause of organic vertigo has been discussed by a number of authors, notably by A. J. Wright,¹¹ and particular attention was directed to this point by Cawthorne, Fitzgerald and Hallpike⁵ in their study of Ménière's disease. The evidence adduced

therein showed that in subjects in whom the diagnosis of Ménière's disease had been made upon the basis of an adequate analysis of symptomatology and physical signs, infective foci in the nose and throat occurred so rarely as to make it difficult to attach thereto any causal significance. In vestibular neuronitis, however, our clinical investigations have led us to the conclusion that infective foci in the nose and throat play an important part in its pathogenesis. This view is based chiefly upon a study of fifty of our cases in whom, in addition to a routine examination of the nose and throat we have also carried out blood examinations including sedimentation rates and radiological examination of the paranasal sinuses. The sedimentation rate tests were carried out according to the Westergren technique. We have regarded as pathological, values for the first hour which exceed 8 mm in the male or 10 mm in the female.

Our 50 cases fall into the following three main divisions with certain sub-groups:

DIVISION I

CASES PRESENTING DIRECT EVIDENCE OF AN ACTIVE INFECTIVE FOCUS

Group A. Antral infection confirmed by the presence of pus on proof puncture. Number of cases, 10. In 4 of these the sedimentation rate was raised.

Group B. Sore throats with obvious evidence of active tonsillar infection. Number of cases, 2. In both, the sedimentation rate was raised.

DIVISION II

CASES PRESENTING STRONG PRESUMPTIVE EVIDENCE OF AN ACTIVE OR QUIESCENT INFECTIVE FOCUS

Group A. Definite radiological evidence of antral infection. Number of cases, 6. In some, proof puncture was refused; in others it was negative. In none of these was the E.S.R. raised.

Group B. Dental infection. Number of cases, 4. The E.S.R. was raised in one case.

Group C. Cases presenting no evidence of infective focus, but giving a clear history of an infective illness at the time of onset of the vertigo. Number of cases, 13. In three of these the sedimentation rate was raised.

DIVISION III

CASES PRESENTING NO EVIDENCE OF AN INFECTIVE FOCUS,
OR A HISTORY OF INFECTIVE ILLNESS

Number of cases, 15. In 4 of these the sedimentation rate was raised.

Our opinion, based upon these findings, that infective processes play an important part in the pathogenesis of vestibular neuronitis, is chiefly derived from the two groups of cases comprised in Division I. From these we see that clear evidence of antral or tonsillar infection was present in 24% of our 50 cases. This far exceeds any corresponding figures that we could base upon our experience of Ménière's disease. The same can be said of Division II, from which we see that 46% of our 50 cases either gave a clear history of an infective illness at the time of onset of the vertigo, or else exhibited significant evidence of an infective focus in the nose and throat. In the other 50 of our 100 cases our examinations for evidence of focal infection have been less complete; that is to say, systematic sinus x-rays and blood examinations have not been carried out. Nevertheless, in these remaining 50 cases we have observed clear evidence of antral infection in 8, and in 14 others there was history of an infective illness at the time of the onset of the symptoms.

(2) *The galvanic test results and their localising value.* The use of the galvanic tests in vestibular neuronitis would appear to be particularly appropriate since the work of Huizinga,¹² Dohlman¹³ and others has suggested that the galvanic responses depend upon the integrity of Scarpa's ganglion and the vestibular neurones central thereto, and are preserved in lesions of the peripheral sense organs. We have, therefore, carried out systematic galvanic tests in a number of our patients according to the procedure previously described in 'Brain.'¹⁴ Details of the test procedure are given as follows:

"Bipolar stimulation was used, the current being passed between brine-soaked pads firmly located upon one or other of the mastoid processes and the manubrium sterni. The tests were carried out with the patient standing with eyes closed and the feet and heels close together. The reaction was described as positive when swaying, which normally occurs towards the ear carrying the positive electrode (anode) could be clearly and repeatedly observed. Corresponding observations were also carried out with the polarity reversed; in these circumstances, of course, the direction of the swaying was also reversed.

"To begin with, a series of control observations was carried out upon twelve normal individuals with no history of ear disease or vertigo. In all of these it was found possible to observe positive responses with values of current lying within the range of 0.3 to 1.9 ma, with an average of 0.8 ma."

TABLE III.—12 NORMAL CASES.

CASE NO.	EAR STIMULATED			
	RIGHT		LEFT	
	POLARITY		POLARITY	
	-VE	+VE	-VE	+VE
1	0.6	0.3	0.6	0.6
2	0.5	0.45	0.5	0.45
3	0.5	0.5	0.4	0.6
4	0.5	0.7	0.5	0.7
5	1.0	1.0	0.5	1.2
6	0.45	0.4	0.45	0.4
7	1.4	1.5	1.4	1.5
8	1.0	1.3	1.0	1.4
9	0.5	0.7	0.5	0.7
10	0.45	0.4	0.45	0.4
11	0.6	0.6	0.55	0.55
12	1.8	1.9	1.7	1.8

Full details of these findings are given in Table III, while the values obtained in 16 cases of vestibular neuronitis are given in Table IV. In both tables the figures give are for milliamperes of current at threshold. The maximum currents given in Table IV were determined by the onset of the usual painful sensations from the skin areas underlying the electrodes.

It will be seen that in all, except three cases, a significant reduction of the galvanic responses was present, a finding which is certainly suggestive of a lesion of the vestibular neurones involving either Scarpa's ganglion, or the vestibular neurones central thereto. It must be added, however, that the mechanism of the galvanic responses is in some ways obscure, and we have observed their derangement both in cases of severe and long-standing Ménière's disease and in certain other disorders in which it might be supposed that the vestibular end organs are chiefly or solely involved. This finding would be explained upon the reasonable supposition that the response to galvanic stimulation is mediated at least in part by the peripheral sense organs. If these are eliminated by disease, then the responses will be reduced but not abolished. The matter is further complicated

TABLE IV.—16 CASES OF VESTIBULAR NEURONITIS.

CASE NO.	EAR STIMULATED					
	RIGHT			LEFT		
	POLARITY -VE	+VE	MAX. CURRENT USED	POLARITY -VE	+VE	MAX. CURRENT USED
1	Absent	3.0	4.0	1.3	1.2	
2	Absent	Absent	5.0	Absent	Absent	5.0
3	1.4	1.4		0.6	0.6	
4	0.7	0.7		3.0	3.0	
5	1.6	1.6		1.4	1.4	
6	2.0	3.0		3.0	3.0	
7	Absent	Absent	3.0	Absent	Absent	3.0
8	1.2	1.5		1.2	1.5	
9	1.0	3.0		4.0	1.0	
10	Absent	Absent	5.0	Absent	Absent	5.0
11	Absent	1.5	3.0	1.5	Absent	3.0
12	0.5	1.0		0.7	0.6	
13	Absent	Absent	2.2	Absent	Absent	2.2
14	1.0	4.0		5.0	5.0	
15	Absent	Absent	5.0	Absent	Absent	5.0
16	0.6	0.6		1.0	1.0	

by the fact that long continued and heavy sedation is likely, in cases of Ménière's disease, to reduce the sensitivity of the central vestibular elements. For this reason, too, one would expect, in long-standing cases of Ménière's disease, to encounter some reduction of the galvanic responses. The subject, however, is a difficult one and clearly calls for further examination. So far, however, as we have considered the matter, it would seem clear that the reduction of the galvanic responses in vestibular neuronitis exceeds in degree what we have encountered in Ménière's disease, and therefore supports our hypothesis of a central affection of the vestibular neurones.

We come, finally, to a third variety of organic vertigo presenting as its chief clinical feature a highly distinctive type of positional nystagmus. This again is distinguishable on clinical grounds alike from Ménière's disease and from vestibular neuronitis. Further, we

have good reason to believe that its pathological basis is also quite different from those of these two conditions. We shall consider it in some detail.

POSITIONAL NYSTAGMUS

This strange and dramatic disorder was first described by R. Bárány in 1921.¹⁵ Bárány mentions two distinct conditions, one of which he attributed to otolith disease. This we shall describe as Bárány's first type. His second type, to which he referred very cursorily, he seems to have attributed to a lesion within the central nervous system.

Bárány had not a great deal to say about either of these conditions, but what he did say was very much to the point, and in due course we shall return to his own words upon the subject.

Since his time many papers have been written about it, short, long and in many languages. Nylén's clinical and animal studies are well known, in particular his monograph on positional nystagmus occurring in intracranial tumours,¹⁶ and in a recent survey of the subject¹⁷ he gives a bibliography of no less than 297 papers written by 192 authors.

Considerable difficulties have been encountered in the preparation of the present communication; difficulties which arise in the main from certain serious deficiencies in the terminology of the subject which now pervade its extensive and confusing literature. Thus, it now seems to be generally agreed that a certain type of positional nystagmus which Nylén defined as the "position changing" type is associated with posterior fossa lesions. This type of positional nystagmus is characterised by the fact that its direction changes when the position of the head is reversed, and it is proposed to exclude it from the present communication. But Nylén also defined another type of positional nystagmus as the "direction fixed" type. In this, certain positions of the head produce nystagmus, and its direction does not change with changes in the position of the head.

Now this, in some respects, corresponds more closely both with what Bárány observed in his case of otolith disease, and with what we have observed in the group of cases now to be described.

It is found, however, on proceeding to this task, that the characteristics of the nystagmus in this, as indeed in any variety of positional nystagmus, cannot be adequately specified in terms of its direction fixation. Furthermore, such a classification takes no note at all of certain other features of the nystagmus which were men-

tioned very clearly by Bárány and are obviously of great importance; so much so that they must inevitably be taken as our starting point. They are as follows: Firstly, the character of the nystagmus which is essentially paroxysmal; secondly, the course of the disease which is essentially benign. It is seldom if ever, we find, associated with any evidence of intracranial disease, and tends to recover with time and simple sedative measures. As will be seen, our evidence goes to show that it is due, as Bárány believed of his case, to a non-progressive lesion of the otolith apparatus. Bárány's own words upon his case may now be quoted. The patient was a 27-year-old woman who had had attacks of vertigo for 14 days. Hearing was normal, the caloric reactions normal, and the central nervous system normal. Bárány writes:

"My assistant, Dr. Carlefors, first noticed that the attacks only appeared when she lay on her right side. When she did this, there appeared a strong rotatory nystagmus to the right. The attack lasted about 30 seconds and was accompanied by violent vertigo and nausea. If, immediately after the cessation of the symptoms, the head was again turned to the right, no attack occurred, and in order to evoke a new attack in this way, the patient had to lie for some time on her back or on the left side."

Bárány goes on to say that similar observations have been made by himself and others, and the re-action had been attributed to lesions of the semicircular canals. In this case, however, Bárány carried out certain further observations, and demonstrated that the factor precipitating the vertigo was not head movement but head position in space, and for this reason he attributed the condition to a disorder of the otoliths.

Our approach to the study of this condition, of which we have now seen a large number of cases, has been primarily clinical. First comes the matter of symptomatology and in few conditions is careful history-taking of such decisive importance. Apart from the patient's account of his symptoms other points of interest are sex and age incidence, relationship of the symptoms to head injury, to focal infection and to collateral evidence of aural or neurological disease. Finally, note is taken of the duration of the symptoms and their response to treatment.

An otological examination is then carried out with full functional tests of hearing and equilibrium, and lastly an examination is made for positional nystagmus.

Symptoms: The story given by the patient is characteristically that the giddiness comes on when he lies down in bed or when he

turns over in bed, or when such a position is taken up during the day; for instance, in lying down beneath a car or in throwing the head backward to paint a ceiling. The patient sometimes, although not always, recognizes that the onset of the vertigo is associated with this critical position and will say that he does his best to avoid it. He may sometimes also say that he has noticed the phenomenon of adaptation which Bárány described so well in his patients, and can cause his vertigo to disappear by maintaining his head in the disagreeable position, or by taking up this position slowly. The vertigo is essentially transient and it is generally accompanied, but not always, by nausea and, it may be, by vomiting. Cochlear symptoms are generally absent; one other symptom of interest is of discomfort, and it may be of tenderness in the occipital region. Examination of the ears, nose and throat reveals in many cases normal findings, and the same can be said of the usual tests of cochlear and vestibular function. These will be considered after first describing the positional nystagmus and our technique for eliciting it.

The reaction is induced, as Bárány said, by a critical position of the head in space. This can be defined as follows: The patient is laid supine upon a couch with his head just over its end. The head is then lowered some 30° below the level of the couch and turned some 30° to 45° to one side. In taking up this position, the patient is first seated upon the couch with the head turned to one side and the gaze fixed upon the examiner's forehead. The examiner then grasps the patient's head firmly between his hands and briskly pushes the patient back into the critical position. The reaction which results calls for some detailed description.

First of all there is nearly always a marked latent period. Sometimes this is as long as five or six seconds. Occasionally it is very short and indeed the reaction may seem to come on at once. This, however, is uncommon. The onset of the nystagmus is nearly always preceded by an appearance of distress. The colour may change; the patients may close their eyes, cry out in alarm and make active efforts, to sit up again. At this point it is necessary to reassure the patient and maintain the position of the head. The nystagmus is chiefly rotatory, the direction of the rotation being towards the undermost ear. (Note—In specifying the direction of the rotation reference is made to the displacement of the 12 o'clock point of the corneal circumference.) In addition to the rotatory element there is generally a horizontal component which is again directed towards the undermost ear. The nystagmus increases in a rapid crescendo in a period which may be as short as $2/3$ seconds, or as long as 10 seconds. Thereafter it rapidly declines and the patient's distress is relieved. If the patient

is then allowed to sit up, a recurrence of the vertigo in a slighter form is generally noted, and if the eyes are examined at this point nystagmus can be seen, the direction of which is, on the whole, reversed. If this is allowed to disappear and the critical supine position is again assumed, the nystagmus again makes its appearance but generally in slighter form and disappears more rapidly than before. After two or three repetitions of this test it is generally found that the reaction has been eliminated altogether and cannot be elicited except, as Bárány pointed out, after a period of rest.

A more detailed consideration will now be given to the oto-rhino-laryngological findings, and the tests of cochlear and vestibular function. A substantial body of our patients, more than a third, have been found to have entirely healthy ears, noses and throats. Tests of cochlear function have been normal; tests of vestibular function, Romberg, gait and caloric responses have also been normal. Rather more than half of the subjects have exhibited substantial evidence of ear disease. This evidence has generally taken the form of tympanic changes indicative of old and sometimes active catarrhal or suppurative otitis media. In a few cases a severe high-tone deafness has been present alone; in some, due to trauma, in others of obscure etiology. In most of these, substantial abnormalities of cochlear function, or of the caloric test results were present. In a good many of these cases with ear disease, one ear alone was affected and, as will later be seen, it has been possible to apply this finding to a solution of the problem of localization of the lesion. On the whole, however, it is true to say that evidence of ear disease, although present in some cases, is inconspicuous in the majority and entirely absent in over a third.

We come, finally, to the course of the disease and the association of the nystagmus with other evidence of neurological disorder. In a good many subjects pain in the neck and occipital region is complained of, and radiological examination has generally revealed some evidence of cervical arthritis. The region affected, however, is chiefly that of the fifth and sixth cervical vertebrae, a very common site in subjects of our predominant age groups, and it has therefore been impossible to attribute any significance thereto. All of our cases have been investigated by our neurological colleagues and, with one or two exceptions of doubtful significance, no evidence has been found of any neurological lesion. The course of the disease is essentially benign. Many of our cases have been followed up for five years or more and in nearly all the symptoms have subsided with sedatives. In a few, infective lesions of the antra or teeth have been

present, and these have been eliminated by appropriate surgical measures. It appears likely that the incidence of such infective lesions in our cases has been abnormally high; certainly more so than in cases of Ménière's disease. The evidence for this is, however, inconclusive and it is certainly true to say that in many cases no evidence of infection has been found.

In reviewing the clinical data so far presented it can be said of this disorder that a great deal is now known of its symptomatology and natural history. Much is also known of its physical signs. It is of interest, therefore, to review this knowledge and consider the information which it yields upon the nature of its pathology.

Two things are quite clear. Firstly the pathological process, wherever or whatever it is, is essentially a benign or self-limiting one. Secondly, the lesion, whatever its nature, is entirely limited to the vestibular apparatus, and here the term 'vestibular' is used in its widest sense, to include the labyrinth, vestibular nerve and its central connections. The questions, therefore, that remain are these: Where exactly is the lesion, and what is its nature?

These we have endeavored to answer by means of further clinical studies, and by the histological examination of the labyrinth in a human subject.

The clinical evidence will be considered first. The benign character of the disorder and the fact that its manifestations are entirely vestibular exclude at once the possibility that it is due to any destructive lesion, vascular, neoplastic, inflammatory or degenerative within the brain stem or VIIIth nerve. If it were, then the occasional occurrence of involvement of other nervous pathways within the brain stem which lie close to the vestibular neurones would be inevitable. Furthermore, some evidence of the destruction of the vestibular neurones themselves would be apparent as, for instance, changes in the caloric test results which are so characteristic of vestibular neuronitis and which we have attributed to a toxic destruction of the vestibular neurones within the brain stem.

It is conceivable, of course, that the nystagmus might be due to some temporary vascular disorder of the brain stem dependent upon some vascular abnormality. Here it is well known that De Kleyn¹⁸ suggested this very possibility and brought forward good anatomical evidence that in some cases at least an abnormality of one vertebral artery rendered it susceptible to occlusion by certain head positions of the very kind that we employ for eliciting the typical reaction of positional nystagmus. We have been in the habit,

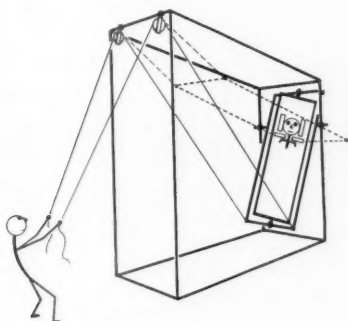


Fig. 7

from time to time, of examining typical cases without any neck-twisting, and recently we have adopted even more elaborate precautions to exclude this possible source of artefact, if such it could be called.

By means of apparatus shown in Fig. 7 it is possible to move the patient *en masse* into the critical position, and under these conditions the characteristic nystagmic reaction has been found to make its appearance as usual. It still remains conceivable that some kind of reversible lesion of the central vestibular neurones due to infection or injury, with or without some anatomical aberration, might bring about the typical reaction to posture now described. Nevertheless, the very fact that the condition never exhibits evidence of any extra vestibular involvement, together with the absence of any evidence of destruction of vestibular neurones, as for instance changes in the caloric responses, makes the possibility of a central lesion exceedingly remote, and on this purely clinical evidence alone we have been inclined to localize the lesion more peripherally and further to believe that it has an essentially irritative basis.

Two questions of great interest now arise: Is the lesion in the labyrinth at all? If so, which is the affected labyrinth? And here it has been possible to advance our ideas very considerably by the further analysis of the clinical evidence.

The total number of our cases is 100. Age and sex incidence are given in Table V. The cases have also been divided into three groups in a manner designed to establish the rôle, nature and localization of ear disease as a possible cause of the positional nystagmus. The analysis is presented in Table VI.

TABLE V.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE. 100 CASES.

Age (Years)	AGE DISTRIBUTION						Total
	20-29	30-39	40-49	50-59	60-69	Over 70	
Number	4	18	27	33	13	5	100

SEX DISTRIBUTION			
Male	52	Total	100
Female	48		

TABLE VI.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE. THE ROLE OF EAR DISEASE AS A CAUSATIVE FACTOR.

	NUMBER OF CASES
GROUP I	
No evidence of ear disease	34
GROUP II	
Slight evidence of ear disease (abnormality of caloric responses only)	11
GROUP III	
Substantial evidence of ear disease (gross middle ear infections, labyrinthine trauma, etc.)	55
Total	100

In the first of these three groups there are 34 cases with no evidence whatsoever of disease of either ear; this group, therefore, provides no evidence of localization. In the next group of 11 cases slight evidence of ear disease has been present, and in the majority of these the disease was unilateral. In the third group of 55 cases the evidence of ear disease was more obvious and again, in a large proportion of cases, the disease was unilateral. Correlation of this evidence of ear disease in these two last groups of cases with the direction of the nystagmus, leads to two important conclusions: 1. that ear disease does play an important rôle in the causation of the nystagmus; 2. that the side of the ear disease is related to the direction of the nystagmus in a systematic manner.

Going back to Group 1: In 31 of these 34 cases the positional nystagmus was unilateral, directed towards the undermost ear. In the other 3 cases, the positional nystagmus was bilateral and again directed towards the undermost ear. It is proper to say of these 34 cases that no evidence is provided as to the localisation of the lesion responsible for the nystagmus.

Further, that the lesion, if it is in one or other labyrinth, has caused no other demonstrable disturbance of its function.

In all 11 cases of Group II the only evidence of ear disease was an abnormality of the caloric test responses. In four of these the caloric abnormality was bilateral. In all four the positional nystagmus was unilateral and directed towards the undermost ear. In all of the other seven cases, in which the abnormality was unilateral, the positional nystagmus was also unilateral and directed towards the side of the caloric abnormality when this was undermost. It is necessary to comment on the fact that in all of the 7 of these 11 cases in which an aural lesion was present, even in the form of a caloric abnormality, the positional nystagmus was always directed towards the affected side when this was undermost. This strongly suggests that the lesion responsible for the positional nystagmus was located within the undermost ear.

We come finally to Group III. The cases in Group III consist of 55 subjects with substantial evidence of ear disease. This evidence is analyzed in Table VII.

The 31 cases in which the disease was bilateral can be put aside as presenting no evidence of localization. Twenty-four cases are left in which the ear disease was unilateral. It will be seen that in no less than 21 of these the direction of positional nystagmus was towards the diseased ear when this was undermost. This evidence

TABLE VII.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE.

GROUP III		DIRECTION OF POSITIONAL NYSTAGMUS	
55 CASES WITH SUBSTANTIAL EVIDENCE OF EAR DISEASE		TOWARDS AFFECTED EAR WHEN UNDERMOST	TOWARDS AFFECTED EAR WHEN UPPERMOST
	BILATERAL	UNILATERAL	
A. Evidence of Otitis Media (Suppurative or Severe Catarrhal)	11	15	15 0
B. Evidence of Neuro-labyrinthitis (Mumps or Syphilis)	1	2	0 2
C. Evidence of Inner Ear trauma	12	6	5 1
D. Other evidence of Ear Disease (Nerve deafness of obscure aetiology)	7	1	1 0
	31	24	21 3

demonstrates again that ear disease is an important factor in causing the nystagmus. Furthermore, that the nystagmus is directed towards the side of the lesion when this is undermost.

We are thus directed to the conclusion that the lesion *is* a peripheral one and in the labyrinth towards which, when undermost, the nystagmus is directed. Consideration may next be given to the nature of the lesion, and here again clinical studies have proved of decisive importance. The benign course of the disorder makes a neoplastic lesion impossible. The usual absence of cochlear dysfunction and the entirely different character of the vertigo differentiates it completely from the typical hydrops of Ménière's disease, and the remaining possibility is thus of a chronic lesion due to infection, trauma or vascular disease. The lesion certainly affects the otoliths and, since it is so often associated with normal caloric responses, it follows that it is more likely to be irritative in character than destructive.

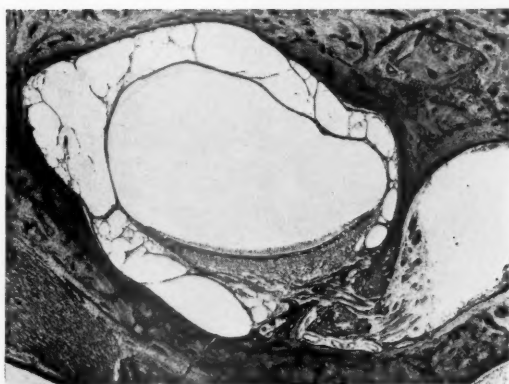


Fig. 8

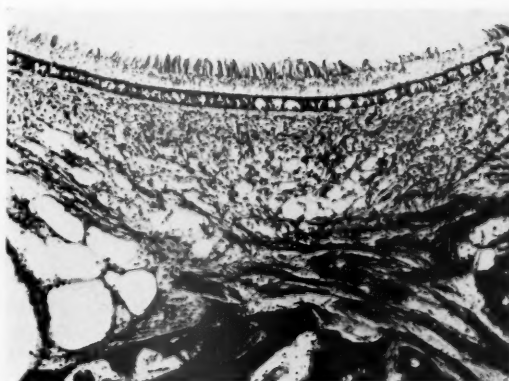


Fig. 9

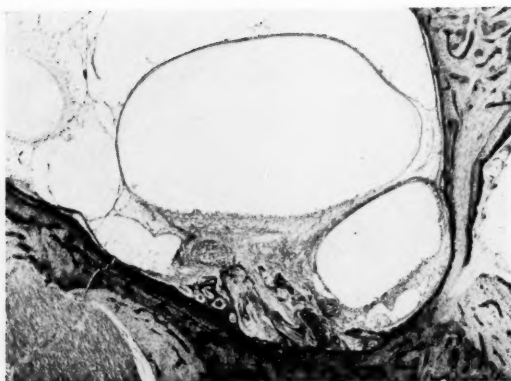


Fig. 10

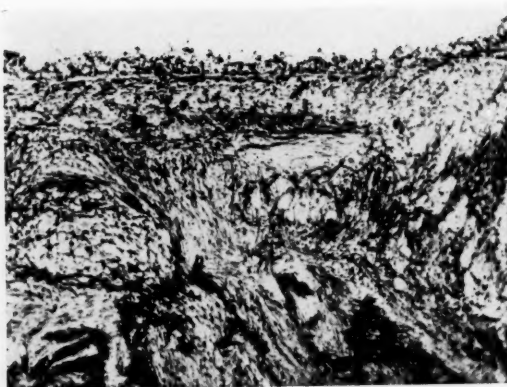


Fig. 11

This concludes our clinical evidence bearing upon the nature and localization of the lesion responsible for positional nystagmus of the benign paroxysmal type.

We have been able to derive supporting evidence from the histological examination of the labyrinths of a characteristic case and an account of our findings is appended.

The case was that of a woman aged 40, a patient of Sir Charles Symonds, who died at the National Hospital, Queen Square, on December 3rd, 1947. For twenty years she had suffered from vertigo with deafness of unknown cause of the right ear. Her terminal illness and death were due to a glioma affecting the basal ganglia and the upper part of the brain stem on the left hand side. On examination a few months before death there was a severe deafness of the right ear of the inner type without evidence of tympanic disease. The caloric responses were brisk and normal on both sides and a positional nystagmus of the benign paroxysmal type was present to the right with the right ear undermost. Her symptoms under these conditions reproduced the vertigo from which she had suffered for twenty years. On histological examination the left labyrinth was normal. The right labyrinth showed a severe degeneration of the spiral ganglion in the cochlea which was the essential cause of the deafness. The ampullae of the semi-circular canals appeared normal. In the maculae of the utricle and saccule, however, very unusual changes were present, in particular the utricle.

In Figure 8 is shown, for comparison, the appearance of the normal, healthy human utricular macula. The layer of sensory cells is seen evenly arranged with the superimposed otolith membrane. Beneath the layer of sensory cells lies a loose connective tissue meshwork, in which run the fibres of the utricular nerve. In Figure 9 is shown a view at higher magnification of the sensory epithelium and the underlying connective tissue meshwork.

In Figure 10 is shown a view of the macula of the utricle in our case of positional nystagmus. The outstanding feature is the absence of the otolith membrane, the disorganisation of the sensory epithelium and certain gross tissue changes in the connective tissue meshwork underlying the epithelium. Its depth is greatly increased. A certain amount of new bone formation has taken place, and at certain points a considerable cellular infiltration is present. This is better seen at higher magnification in Figure 11 where it can be seen that in addition to the absence of the otolith membrane and the disorganisation of the sensory cells, there is also present a considerable thickening of the sub-epithelial connective tissue network with the presence here and there of irregular cellular infiltrations. At one point there occurs a number of irregular spaces occupied by fluid or cell remnants. The general picture is one of chronic tissue changes resulting either from infection or trauma and it accords very well with our conception of the responsible lesion which we have reconstructed from our clinical evidence. Changes very similar in character but lesser in

degree were present in the macula of the saccule. This concludes our clinical and pathological evidence on the subject of this variety of positional nystagmus. As already stated, it would seem essential to describe it as the benign paroxysmal type on account of certain outstanding clinical features which distinguish it just as much as its directional characteristics.

In order to achieve clarity, emphasis has been laid in the course of this communication upon such facts—on the whole many and weighty—which fortify the central theme of our argument. Certain discrepancies exist; they have not been overlooked and will be given attention in the course of further studies. We do not think that they will be found to invalidate our main conclusion which is that positional nystagmus of the benign paroxysmal type, first described by Bárány in 1921, is due, as Bárány believed to otolith disease. The lesion consists of chronic tissue changes which may be due to trauma, chronic infection or possibly to vascular disease. It affects, and may be confined to, the sensory epithelium and the subepithelial connective tissue of the utricle and saccule of the labyrinth towards which, when undermost, the positional nystagmus is directed.

It is a pleasure to acknowledge the great help that we have received in the course of these investigations from our colleagues, the physicians and surgeons of the National Hospital, Queen Square, who have so kindly put their cases at our disposal; in particular, to Sir Charles Symonds, two of whose cases have proved of special importance; also to Dr. C. H. Edwards, who gave us considerable help in the early stages of the preparation and analysis of the clinical findings of our cases of positional nystagmus. Finally, for the construction of the apparatus shown in Fig. 7 and of other special test equipment, and for the preparation and photography of the histological material we are greatly indebted to the laboratory staff of the Otological Research Unit of the Medical Research Council.

REFERENCES

1. Hallpike, C. S., and Cairns, H.: *Jl. Laryng. and Otol.* 53:625, 1938.
2. Day, Kenneth M.: *Trans. Amer. Otol. Soc.* 38:23, 1950.
3. Wells, Walter A.: *Laryngoscope* 57:275, 1947.
4. Williams, Henry L.: *Trans. Amer. Otol. Soc.* 37:76, 1949.
5. Cawthorne, T. E., Fitzgerald, G., and Hallpike, C. S.: *Brain* 65:161, 1942.
6. Dix, M. R., Hallpike, C. S., and Hood, J. D.: *Proc. Roy. Soc. Med.* 41:516, 1948.
7. Hood, J. D.: *Proc. Roy. Soc. Med.* 43:1129, 1950.
8. Hallpike, C. S.: *Proc. 4th Int. Cong. Otolaryngol.* 2:514, 1949.
9. Fitzgerald, G., and Hallpike, C. S.: *Brain* 65:115, 1942.
10. Hallpike, C. S., Harrison, M. Spencer, and Slater, Eliot: *Acta Otolaryng.* 39:151, 1951.
11. Wright, A. J.: *Jl. Laryng. and Otol.* 53:97, 1938.
12. Huizinga, E.: *Acta Otolaryng.* 15:451, 1931.
13. Dohlman, G.: *Acta Otolaryng.* 26:425, 1938.
14. Dix, M. R., Hallpike, C. S., and Harrison, M. Spencer: *Brain* 72:241, 1949.
15. Bárány, Robert: *Acta Otolaryng.* 2:434, 1921.
16. Nylén, C. O.: *Acta Otolaryngol. Suppl.* 15, 1931.
17. Nylén, C. O.: *Jl. Laryng. and Otol.* 64:295, 1950.
18. De Kleyn, A. u. Nieuwenhuyse: *Acta Otolaryng.* 11:155, 1927.

MASSIVE OSTEOMA OF THE ETHMOID SINUS

MARVIN J. TAMARI, M.D.

AND

EDWARD B. WEISMAN, M.D.

CHICAGO, ILL.

Osteomas are the most common benign tumors of the skull. Our unusual experience in finding a massive asymptomatic tumor of the ethmoid sinus prompted us to report this case.

CASE REPORT

On August 22, 1951, M. O., a 61 year old colored female was seen at the Illinois Eye and Ear Infirmary for a complaint of bilateral nasal discharge since the age of 10.

The onset of a yellow-brown mucoid nasal discharge was associated with an episode of trauma, (a boy hit her on the nose without any injurious effects). No watery nasal discharge, bleeding or unconsciousness occurred. In the past 50 years she was seen by two physicians and given only local nasal therapy. Since 1939, she noted an impaired sense of smell.

As a child, she had measles, mumps and whooping cough. No other medical or surgical problems.

On physical examination this woman was short, stockily built and appeared younger than her chronological age. Pathological physical findings were: bilateral pale polypoid masses of the mucosa occluding all but the inferior meatuses of the nasal fossa, polypoid swelling of the anterior end of the left inferior turbinate, mucopus covering the polyps, slight septal deviation to the left and slight hypertrophic changes of the posterior tips of both inferior turbinates.

On examination of the eyes no abnormalities were found. Neurologic examination showed all cranial nerves intact, except for the loss of sense of smell to lemon and camphor. An electroencephelo-

From the Department of Otolaryngology, College of Medicine, University of Illinois and Illinois Eye and Ear Infirmary.

**Assistant in Otolaryngology, College of Medicine, University of Illinois and Illinois Eye and Ear Infirmary.



Fig. 1.—Photograph of the patient.

gram study demonstrated 8 to 9 oscillations per second with considerable (30 to 40 per second) activity in the left fronto-parietal area, which was maximal in the pre-central area. No evidence of allergy was found.

On transillumination, the antra and frontal sinuses were cloudy. An irrigation of the left antrum revealed a thick purulent return and lipiodol was instilled for further study.

The initial x-rays taken demonstrated moderate clouding of both antrums and a large density behind the left frontal sinus, extending to the lesser wings of the sphenoids and limited medially at the midline of the anterior fossa and inferiorly invading the ethmoids. Some destruction of the left orbital ridge shadow was suspected on subsequent films. This tumor measured 52 mm x 43 mm x 40 mm on the x-ray films taken at 40 inches from the tube. A stereoscopic x-ray study offered a better appreciation of this mass with its extension into the ethmoid cells. The lipiodol study of the left antrum revealed thickening of the sinus mucosa.

Other laboratory studies revealed R.B.C. 4,390,000, Hemoglobin 87%, W.B.C. 8,250, polymorphonuclear neutrophils 60,



Fig. 2.—Caldwell view of skull.

Fig. 3.—Lateral view of skull.

lymphocytes 39, monocytes 1. Urinalysis normal, Wasserman and Kahn were negative, Blood sugar 63mgm%, Non-protein nitrogen 36mgm%, Blood calcium 9.6mgm%, Blood inorganic phosphorus 2.7mgm%, Alkaline phosphatase 4.1 units per 100 cc of serum, Total serum protein 7.6mgm%, serum albumin 4.8mgm%, serum globulin 2.8mgm%.

On November 20, 1951, a diagnostic exploratory procedure was done through a left eyebrow incision. The skin and periosteum were reflected over the anterior wall of the frontal sinus. With gouge and mallet the anterior wall of the left frontal sinus was removed. The thickened mucosa of the sinus was incised and purulent secretions encountered. The secretions were sent to the laboratory for bacteriologic study. Due to the extensive amount of suppuration, the right frontal sinus was explored, with similar findings. The ostia were left intact. After removal of the exudate no evidence of a pyocoele was found. We proceeded to remove the posterior wall of the left frontal sinus. The dura was seen unassociated with the frontal sinus and covering a solid tumor with no delineation of its extent on inspection. By palpation we noted the tumor to be limited in the midline and covered with dura in its entire anterior ex-



Fig. 4.—Submento-vertex view of skull.

posure. Gently teasing the dura off of the mass from below for a distance of 2 cm we noted the solid bony growth, ivory in color, with pseudolobulated crevices. A specimen was removed for histologic examination. Having explored the tumor and in the presence of suppuration and possible damage to the dura, we were content to do a limited procedure. Iodoform packing was placed in both sinuses, drains being inserted through the eyebrow incisions. Following closure we did a bilateral polypectomy and antrotomy to correct the other pathological processes. The patient had an uneventful post-operative course with resolution of nasal symptoms.

Cultures of the frontal sinus exudate revealed hemolytic staphylococcus aureus and in vitro sensitivity study showed resistance to all of the antibiotics.

The gross specimen of the osteoma consisted of bone chips measuring 1 x 1.2 x 0.2 cm.

The microscopic examination of the decalcified bone as described by one of us (M. J. T.) revealed a hematoxylin eosin stained specimen. Under the thickened and vascularized periosteal layer, there were a few signs of superficial new bone formation in the form of slender osteophytic bony bars found within the periosteum. In the adjacent fibrous marrow spaces surrounded by regular Haversian

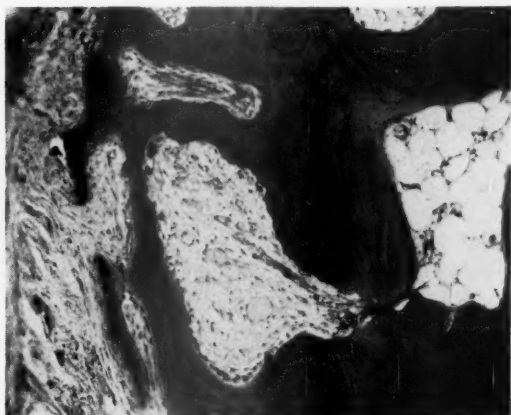


Fig. 5.—Microscopic superficial bone.

systems, bone destruction was observed with typical lacunar absorption by giant osteoclasts. The adjacent deeper bone structure appeared more spongy in character with marrow spaces filled with fat cells and enlarged capillaries but without indications of recent bone activity (Fig. 5).

The biopsied hard tumor represented a bone of denser structure. The pattern of the Haversian system appeared distorted. The marrow spaces, scanty and narrowed. They were filled with loose fibrous tissue and few thin-walled vessels. The osteocytic lacunae in the bone around the marrow spaces were partly empty. Signs of bone activity, such as new bone formation or resorption, were not noted.

In more centrally situated bony spicules, the structure of the bone matrix appeared more amorphous. There were no Haversian systems developed and bony bars were found as prominent elevations into irregularly shaped marrow spaces. The latter contained a few fibrous bands, some enlarged capillaries and extravascular blood. Aplastic surface resting lines and scanty resorption lines pointed to previous bone changes.

The lacunae in the amorphous bone were partly empty and the preserved osteocytes which were present exhibited a shrunken cytoplasm and pycnotic distorted nuclei (Fig. 6).

No histological indications were found to characterize the tumor as a secondary growth from connective tissue or from cartilage and



Fig. 6.—Microscopic central bone.

as such, the tumor was classified as a benign, true osteoma of the ethmoid sinus.

SURVEY

Teed¹ offers us a good historical review of the literature. Of interest was the milestone in medicine, when roentgen rays were developed. Novick² cites Coppez as giving the first case roentgenologically demonstrating the osteoma in 1899. Cushing³ and Hempstead⁴ have utilized stereoscopic roentgen study to more closely evaluate the extent of the tumor. We found this technique of invaluable aid in appreciating this bony configuration. Lillie⁵ described, and we could appreciate, the ramifying characteristics of the osteoma through the ethmoid cells like the pseudopodia of the amoeba.

Childrey⁶ reported a 0.43% incidence of osteomas in his series when routine x-rays were taken. The studies of most authors are also in agreement that the greatest number of osteomas occur in the frontal sinus region and followed by those in the ethmoids. Courville and Crockett⁷ state the osteoma to be the most common tumor of the skull and Eggston and Wolf⁸ cites osteoma to occur in 50% of the benign tumors in the nose and the paranasal sinuses. Ward and Hendrick⁹ consider the head and neck rare sites for primary tumors but more common for metastases. They state that cholesteatomas and hemangiomas follow osteomas in frequency of occurrence.

Lederer¹⁰ and others offer three theories of origin of osteomas: 1) embryologic 2) traumatic 3) infectious. It is current opinion that periosteal rests are the source of development of the osteoma, however, the three theoretical factors are difficult in isolating as to their specific significance in the case reports studied. Fetisoff¹¹ adhered to Echert's work on the embryologic theory of development. Smith considered trauma an exciting factor, although many osteomas may be primary true neoplasms. Ward and Hendrick,⁹ citing Geshichter, support the consideration of trauma in the etiologic role. Then again, Hempstead¹ stated that trauma is insignificant since it is so frequently elicited in the history. Valdala and Somers¹² question the consideration of the infectious etiology and state that it may be of primary, secondary or incidental importance. Childrey⁶ is of the same opinion. It was Ersner and Saltzman¹³ who stated that trauma and infection are exciting causes and offered support of the infectious factor. They referred to the work of Mosher, Green and Shannon to support their hypothesis of infection and the role of the hemolytic staphylococcus albus and opsonic selectivity in creating the bony changes in the involved area.

In surveying size and weights of tumors, the report of Beck¹⁴ refers to a 587 gm osteoma. He adds that such tumors develop because they were considered inoperable. While we cannot offer the weight of this growth, it is the largest asymptomatic tumor we have ever encountered. Its appearance was somewhat like that reported by Wertheimer and Lecuire.¹⁵

Beck,¹⁴ Smith,¹⁶ Hallberg and Begley¹⁷ cite the incidence of growth of osteomas to be encountered most frequently in the early decades of life. The potential malignant behavior of this benign histologic growth is recognized by all authors. The more cellular growths are described as being in the younger individuals and may be termed ossifying fibromas, while in the older group the bone is more compact and may be called osteomas (Eggston and Wolf).

Novick² cites Geschichter and Copeland stating that pathologically the structure elements and the developmental stages of bone are significant in the growth to a mature state. Eggston and Wolf⁸ stated "all bone tumors are essentially mesenchymal and the type cells present may show all stages of metamorphosis. Bone marrow cells and endothelial cells may be present. The osteomata have the histologic characteristics of giant cell tumors and osteitis fibrosa and therefore may have a common cause."

Fetisoff¹¹ citing Eckert (1922) reports that the periosteum invades the mass of the tumor and that growing develops from within

outward. Essentially the osteomas develop from a split off segment of periosteum during a stage of growth of the organism. Brunner and Spiesman¹⁸ describe growth as occurring by metaplastic ossification of connective tissue with deposition of new bone by the paranasal mucosa which acts as a periosteum.

The anatomical origin is listed in relation to the sinus involved and their respective walls. Courville and Crockett⁷ refer to origin in the internal or external tables or diploe. Embryologic consideration of the junction of the intramembraneous frontal bone joining the enchondral ethmoid bone giving rise to cell rests is another site described.

Lillie⁵ pictured the osteomas of the ethmoid as being like pseudopodia of the amoeba which may ramify the ethmoid cells and it is stereoscopy that instills real meaning to this description.

Pathologic classifications vary slightly and Teed¹ offers us the four types of osteomas: 1) Eburnated type having no Haversian canals 2) Compact type with small Haversian canals lying in all directions 3) Spongy type which are softer than the above two with bony trabeculae and intervening marrow spaces 4) Mixed type in which the prepondering type of tissue depends on the age of the growth. The specimens of tumor removed in this case appear to be of the mixed type, which is the most frequently encountered variety.

Many prominent otolaryngologists of this country preoperatively had seen this patient and her films. Their impressions included osteoma, calcified mucocele, pyocele, brain abscess, and hyperostoses due to meningioma. Hallberg and Begley state the osteomas of the ethmoids may be confused with meningioma but are far more dense. Courville and Crockett⁷ evaluate the hyperostosis of the skull due to meningioma and mention the usual location as being in the vertex. Ward and Hendrick⁹ describe the meningioma as also occurring in the basal areas, in both wings of the sphenoids and the temporal squama. We could not help but feel a strong need for exploration in view of the multiple possibilities.

There are two schools of thought with regard to treatment quoted in the last decade. The French School is inclined to remove osteomas whenever found. The English and American surgeons have been more conservative. Our neurosurgical consultant (Dr. Eric Oldberg) was not inclined to remove this growth. We too felt that this was the best course to follow but considered an exploratory procedure valid. Cushing,³ Hoover and Horrax,¹⁹ Lillie,⁵ Hempstead,⁴

Valdala and Somers,¹² Hallberg and Begley¹⁷ offer a variety of surgical approaches to the paranasal sinus osteomata. Hoover and Horrax¹⁹ stated that if the tumor is eburnated one may leave the pedicle, whereas, if the tumor is spongy one must remove some of mother bone or it will recur. Hempstead⁴ advised the fronto-ethmoid approach if infection of the sinus existed and Cushing's osteoplastic flap is the dura and cribriform plate were involved. Petit-Dutaillis and Winter²⁰ considered the rhinological and neurosurgical approaches in their case reported. We chose the eyebrow incision with extensions bilaterally as needed. The suppuration in both obstructed frontal sinuses further increased our limiting the surgical procedure.

A. Eckert Mobius, as quoted by Hoover and Horrax¹⁹ cites a 48% mortality rate from nonoperated osteomata and the latter authors cite a 7 to 10% operative mortality. They add that the osteomas that invade the cranial cavity and go to surgery are the ones with the highest mortality rate. Teed says that the preantiseptic surgical mortality was 31.2% and since 1875 it has been only 3.7%. Hallberg and Begley¹⁷ had no fatalities in their series. In view of the expected behavior of the tumor, the patient's age, x-rays and clinical followup and modern surgical facilities for the geriatric patient, we are satisfied in our conservatism. The patient is of average intelligence and exhibits no apprehension with regard to the tumor's presence. The subject has been discussed with her and she knowingly accepts the medical problem as presented.

SUMMARY

A case is presented of an unusually large true benign osteoma of the ethmoid sinus which had not caused any symptoms in its intracranial or intranasal ramifications.

904 WEST ADAMS.

REFERENCES

1. Teed, R. W.: Primary Osteoma of the Frontal Sinus, *Arch. Otolaryng.* 33:255 (Feb.) 1941.
2. Novick, J. N.: Osteoma of the Frontal Sinuses, *Arch. Otolaryng.* 46:655 (Nov.) 1947.
3. Cushing, H.: Experiences with Orbito Ethmoid Osteomata Having Intracranial Complications, with Report of Four Cases, *Surg., Gynec. and Obst.* 44:721 (June) 1927.
4. Hempstead, B. E.: Osteomas of the Paranasal Sinuses and the Mastoid Process, *J. A. M. A.* 11:1273 (Oct. 1) 1938.
5. Lillie, H. I.: External Operations on the Frontal Sinus, *Am. J. Surg.* 42:199 (Oct.) 1938.

6. Childrey, J. H.: Osteoma of the Sinuses, Frontal and Sphenoid Bone, *Arch. Otolaryng.* 30:63, 1939.
7. Courville, C. B., and Crockett, H. G.: Hyperostosing Osteoma of the Skull, *Bulletin of the Los Angeles Neurological Society* 13-14:86 (June) 1948.
8. Eggston, A. A., and Wolf, D.: *Histopathology of the Ear, Nose and Throat*, Baltimore, Williams and Wilkins Company, 1947.
9. Ward, G. E., and Hendrick, J. W.: *Tumors of the Head and Neck*, Baltimore, Williams and Wilkins Company, 1950.
10. Lederer, F. L.: *Diseases of the Ear, Nose and Throat*, Philadelphia, F. A. Davis Company, 1952.
11. Fetisoff, A. G.: Pathogenesis of Osteoma of the Nasal Accessory Sinuses, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 38:404 (1929).
12. Valdala, A. J., and Somers, K.: Osteoma of the Frontal Sinus, *Arch. Otolaryng.* 50:618 (Nov.) 1949.
13. Ersner, M. S., and Saltzman, M.: Osteoma of the Sinuses, *Laryngoscope* 48:29 (Jan.) 1938.
14. Beck, J. C.: Some Uncommon Types of Neoplasms About the Head: Osteomata of the Frontal Sinus; Multiple Myeloma; Intrapalatal Tumor, *Tr. Am. Laryng., Rhin., and Otol. Soc.* 36:374-386, 1930.
15. Wertheimer, P., and Lecuire, J.: Voluminous Osteoma of Ethmoid Origin occupying the anterior aspect of the base of the cranium, *Lyon Chir.* 45:623 (July) 1950.
16. Smith, A. T.: Osseous Lesions of Nose and Sinuses, *Arch. Otolaryng.* 31:289 (Feb.) 1940.
17. Hallberg, O. E., and Begley, J. W. Jr.: Origin and Treatment of Osteomas of the Paranasal Sinuses, *Arch. Otolaryng.* 51:750 (May) 1950.
18. Brunner, H., and Spiesman, I. G.: Osteoma of the Frontal and Ethmoid Sinuses, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:714 (Sept.) 1948.
19. Hoover, W. B., and Horrax, G.: Osteomas of the Nasal Accessory Sinuses, *Surg., Gyn., and Obst.* 61:821 (Dec.) 1935.
20. Petit-Dutaillis, D., and Winter, P.: Voluminous Osteoma of the Frontal Sinus, *Semaine Hop. Paris* 26:553 (Feb.) 1950.

SOME PRINCIPLES IN THE STRUCTURE OF
VIBRATILE CILIA

H. ENGSTRÖM, M.D.

AND

J. WERSÄLL, M.D.

STOCKHOLM, SWEDEN

Many authors have described the cilia in the animal kingdom, and the great similarity of these organs in different species has been pointed out by Brown,¹ Tremble,² and others. Fundamental, recurrent structural principles are found in the flagella of the primitive Protozoa and in the vibratile cilia of higher animals and of man.

It is evident from the basic studies of de Heide, Sharpey,³ Purkinje and Walentin,^{4, 5, 6} as well as from more recent investigations (such as those of Erhard,⁷ Gray,^{8, 9} Proetz,^{10, 11, 12} Hilding,^{13, 14, 15} Ballenger,^{30, 31} Frenckner and Richtner^{16, 17, 18}) that the cilia activity is a factor of the greatest importance for the organism. Many similar factors are found in the bodily movements of the lowest animals by means of the cilia, and the ciliary activity in for instance, the nasal cavities, the trachea and the bronchi. In all such investigations, considerable difficulties have been encountered in elucidating the mode of ciliary action.

The basic principles of ciliary structure have been studied with the common light microscope. It has been established by a number of workers that, at least in some of the lower animals, the cilium has both an axial, denser part and a thin peripheral sheath. Thus, Awarinzew¹⁹ found an axial fiber in the flagella of the *Chilomonas*. Bütschli²⁰ described such a central ciliary structure in the *Flagellata*. Several other authors, for example Prowazek^{21, 22} and Koltzoff,²³ also distinguished an axial thread and a peripheral sheath in the cilia of some of the lower animals.

Erhard⁷ gave a comprehensive review of the problems; he also discussed other matters of interest with regard to the cilia and ciliary

From the Department of Histology Karolinska Institutet and the Department of Oto-Laryngology Sabbatsberg's Hospital, Stockholm.



Fig. 1.—Bacterium with Single polar cilia. Strain unknown.

action. Electron microscopic studies by Brown¹ provided conclusive evidence in support of the aforementioned observations of an axial core and provided information on other aspects of ciliary structure. Jakus and Hall,²⁴ also using the electron microscope, were able to detect a fibrillar structure in the cilia of *Paramecium*. They did not, however, observe any peripheral sheath surrounding the central fibrils.

On the basis of recent electron microscopic studies, one of us²⁵ (Engström) gave a detailed description of the vibratile cilia of the tracheal cells in some higher mammals and in man. It is apparent from these studies that each tracheal cilium consists of a basal body from which a minute rootlet descends into the protoplasm. A small cuticular plate surrounding the different cells is present at the cuticular border. The outer vibratile part of the cilium consists of an axial bundle of fibrils, usually 11 in number. They form a fairly firm core with high density (against the electron beam). The core is surrounded by a thin sheath of protoplasm with lower density (Figs. 3 and 5).

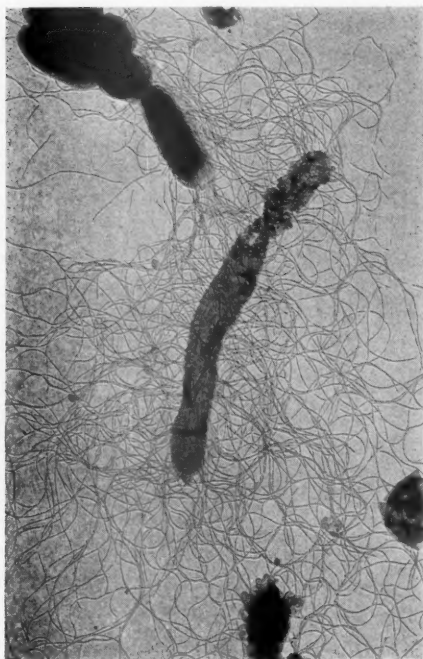


Fig. 2.—*Proteus* bacteria with a large number of flagella. Shadow cast. Magnification about 15 000 x.

The present paper is a report of further studies of the structure of cilia and a discussion of some new factors of interest for the understanding of ciliary activity.

STRUCTURE OF SOME DIFFERENT FORMS OF CILIA

Bacteria. The cilia of the bacteria have been thoroughly studied by a number of authors (*e.g.*, Wyckoff²⁶ and Weibull^{27, 28}) and several different arrangements described. As a rule, they occur as fine threads, singly or several in number, extending from the bacterial body (Figs. 1 and 2). Their incidence, size and properties are now well known. Many workers consider them to be organs of motility; others deny this. Their structure is not, however, similar to that of the vibratile cilia of cellular bodies. For this reason, they will not be discussed further in this paper.

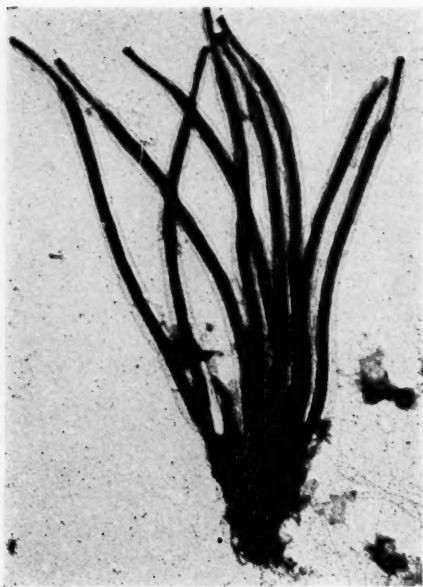


Fig. 3.—Bundle of cilia from the trachea of a dog. The axial core and the thin peripheral sheath are easily recognized. In the central core, the fibrils can be seen. Magnification 23 000 x.

Cilia in the respiratory tract. We have made a particular study of these cilia and have found their appearance to be fairly uniform (Figs. 3 and 8).

A large number of cilia arise from each surface cell in the respiratory tract. Their length varies from about 3-8 microns in the different animals. In man, the tracheal cilium is 6-7 microns long. The number of cilia on each cell is proportional to the size of its free surface. Because a number of cells have a very small upper surface, and giant cells are also found, the number of cilia can only be stated in terms of their occurrence per square micron. We have calculated the number of cilia per square micron in sections of tracheal cells and found it to be around 25-35. Thus, a single tracheal cell with a surface area of 50 square microns would have more than one thousand cilia. These figures are, however, somewhat uncertain. It is nevertheless unquestionable that the cilia are extremely numerous; this is clearly visible under the electron microscope.

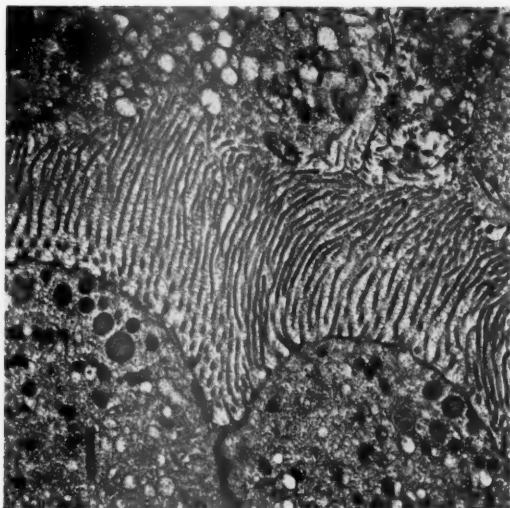


Fig. 4.—Ciliated epithelium from the oviduct (mouse). The cilia are here much smaller and thinner than in the respiratory tract. The structure at the cuticular border is here not distinct. Enlargement about 20000 x. Plastsection.

Each cilium has a basal body situated just below the cuticle of the cell. This body is often flask-shaped and appears to be intimately connected with the cuticle. When the cilia of a cell are separated from each other, a flat collar of cuticle seems to be consistently present immediately above the basal body (Fig. 8). One, or sometimes two, minute rootlets extend into the interior of the cell body. The rootlet is often thicker near the basal body and tapers towards the end. The length appears to vary appreciably in different species. In several higher mammals we were able to follow the rootlet for a distance of $1\frac{1}{2}$ to 3 microns; in some lower animals it was found to be considerably longer.

The outer vibratile part of the cilium consists of an axial fibrillar core and a less dense peripheral sheath (Figs. 3, 4 and 5). The sheath appears at times to have a spiral or helical organization. As, however, this phenomenon was inconstant in our preparations, we are as yet uncertain of its importance. A similar structure in some animals of the lower orders was earlier described by Brown.

The axial core commonly consists of about 11 fibrils, running from the base to the apex of the cilium. They seem to be of approx-

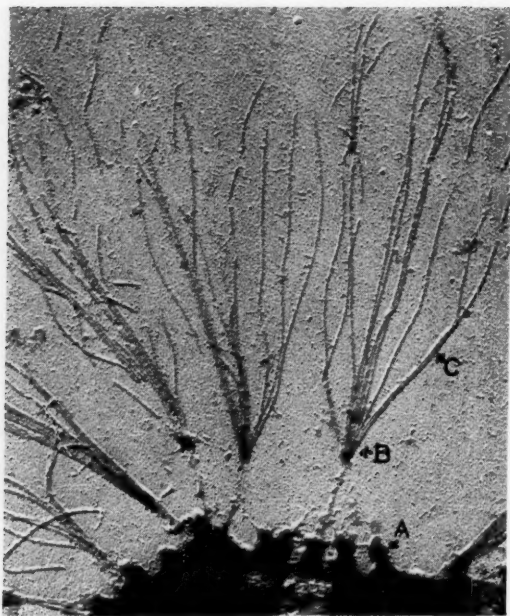


Fig. 5.—Dispersed ciliary fibrils from the avial core after destruction of the peripheral sheath. The picture is indistinct but gives a good idea of the arrangement of the fibrils when they are loosened from the basal bodies. Guinea-pig, trachea. A=Basal bodies, B="Shaft" of the cilium, C=Intraciliary fibrils.

imately the same caliber (300-500 Å in dried preparations). The fibrils are bound together more firmly at the basal and at the apical end. When treated with such methods as freezing or ultrasonics, they frequently separate from each other, except at the base and the apex. We have been unable to find any definite periodicity in these fibers, but it has been pointed out by several authors (e.g., Schmidt²⁹) that protein fibres with a caliber of 300-500 microns are often contractile. It has earlier been observed that the size and number of fibrils in the motile tail of the spermatozoon are amazingly constant, the number ranging from 9 to 12. Jakus and Hall found about 11 fibrils in the cilia of *Paramecium*. We found around 10 fibrils fairly constantly in the tracheal and nasal cilia of the ox, sheep, dog, guinea-pig and frog, and in man. The number of fibrils in the cilia of several Protozoa also appears to be in the neighborhood of 10.

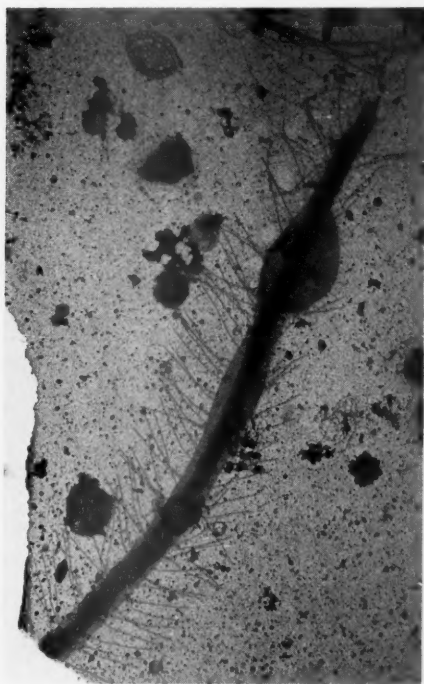


Fig. 6.—"Flimmer" flagellum from a Protozoon. In this case a central core, a peripheral sheath and a brush-like border of "flimmer-hairs" are visible. These hairs can often be seen to project in groups, frequently with four hairs in each group.

Ballenger's^{30, 31} paper is of considerable interest in this connection; it also contains a discussion of a number of structural aspects of vibratile cilia (p. 353).

"Flimmer" flagella. In 1894, Fischer³² described an external structure with fine fibrils extending from the real flagellum in certain Protozoa. These flagella, denoted by him as "flimmer" flagella (Flimmer-geisseln) were for long regarded as artefacts, but later investigations by, among others, Brown,¹ have confirmed their existence and given a relatively reliable picture of their structure.

In order to complete our studies on vibratile cilia, we studied some Protozoa of unknown strains. In their case as well, we distinctly observed an axial fibrillar bundle and a peripheral, less dense sheath. A large number of extremely thin flimmer hairs projected

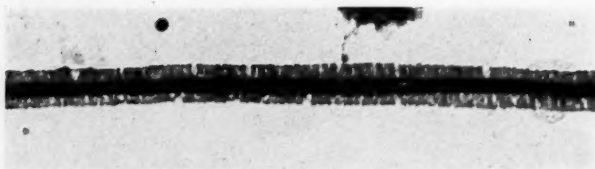


Fig. 7.—Spermatozoan tail consisting of a central fibrillar core and a helical, peripheral sheath. The resemblance to the cilia is evident.

from the central part of the cilium. They were about 1.5μ long, 150 \AA thick and often seemed to be assembled in groups of four.

Spermatozoan tail. Many workers have studied the structure of the spermatozoan tail. Retzius,³³ Ballowitz,³⁴ Bowen,³⁵ and others used the light microscope and the ultraviolet microscope, Schmidt²⁹ polarized light, and Bretschneider,³⁷ Randall and Friedlaender,³⁸ among others, the electron microscope.

It is generally considered that the spermatozoan tail is built up of an axial fibrillar core and a peripheral, less dense, helical sheath. Statements regarding the number of central fibrils have varied, figures up to 12 double fibrils having been given (Randall and Friedlaender). The caliber has been found to vary in different types of fibrils. Electron micrographs of the spermatozoan tail of the rabbit distinctly show an outer helical sheath and a number of central fibrils (Fig. 7). There is thus a general similarity between the structure of this tail and that of the vibratile cilia of the respiratory tract. Moreover, both of them are mobile. Some authors have, in fact, expressed the opinion that the spermatozoan tail may be regarded as a specialized cilium.

DISCUSSION

An investigation has been made with the electron microscope of the organization of different flagellar structures. The flagella of bacteria have been found to differ from those of cellular organisms and to consist of fibers of varying length, diameter and number. On the other hand, a striking structural resemblance is found between the flagella of many cellular organisms; this applies to the vibratile cilia of several Protozoa, sauroids and mammals.

Thus, in the Protozoa and in the vibratile cilia from the pharynx of lizards and frogs and the respiratory tract of mammals, we often found a central fibrillar core of fairly high density (against the electron beam) and a peripheral sheath of lower density. In mam-

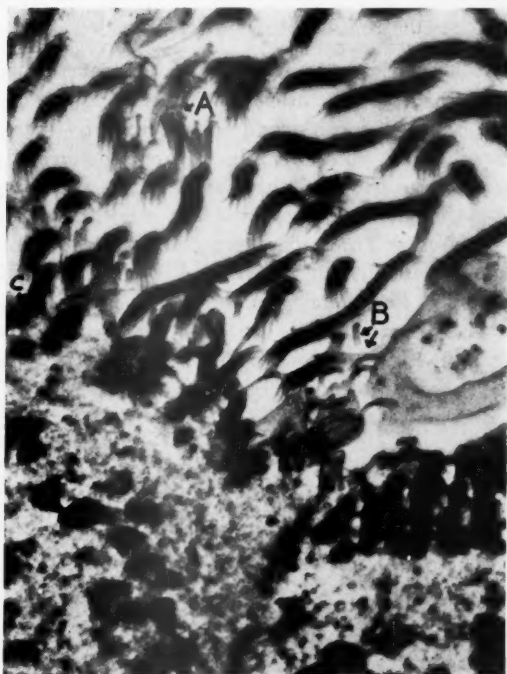


Fig. 8.—Section from a tracheal cell. Guinea-pig. In the lower part of the picture the interior of the cell can be seen and at A a cross-cut cilium with the typical arrangement of the central fibrils. At B the inter-ciliary processes and at C the cuticular plate. Plast. section. Veronal-buffered osmic acid fixation. Magnification about 20 000 x.

malian vibratile cilia, the central fibrillar core consists of a fairly constant number, usually 11, of fibrils; their caliber is also relatively uniform, *i.e.*, 300-500 Å. Cross sections of tracheal cilia clearly show that the central fibrils are almost invariably arranged with two fibrils in the center, surrounded by nine fibrils in the form of a ring (Fig. 8). In primitive cellular organisms as well, the number of fibrils is often similar; according to Jakus and Hall, it is around 11 in *Paramecium*. In addition, a number of authors have stated that the spermatozoan tail possesses 11-12 fibrils.

The structural organization of many motile flagella and cilia thus shows agreement with regard to the vibratile part. A question of prime importance is therefore whether the contractile ability of these motile parts is localized in the central fibrillar portion or in

the sheath. A study of the chemical composition of vibratile cilia is necessary before this problem can be solved.

In the cilia of multicellular organisms, one or several basal bodies are usually present below the cuticle of the cells. These bodies have been the object of much interest and are regarded by many authors as the center of ciliary motility. In the present investigation, a careful study has been made of these basal bodies and a number of facts relating to their structure reported. It is not, however, possible to draw any inferences from them regarding their possible significance for ciliary motility. Of importance in this connection is the fact that, in several Flagellata with motile flagella, no basal bodies are present. Their central fibrils are considered to terminate freely in the interior of the cell. We are at present making further studies on these and related problems of ciliary development, the chemical composition of the ciliary sheaths and the influence of enzyme digestion.

SUMMARY

The structure of vibratile cilia is discussed and the following observations made from electron microscopic studies:

1. Bacterial cilia or flagella consist of thin fibrils of varying length and diameter. These flagella differ from the vibratile cilia of many cellular organisms.

2. Vibratile cilia of several Protozoa, sauroids and mammals show a striking structural similarity. Thus, there is often a basal body with at least one minute rootlet, and a vibratile portion above this body. The vibratile part frequently consists of a central fibrillar core, surrounded by a fragile, sometimes helical, sheath.

3. The fibrillar, central part commonly comprises about 11 fibrils, running the whole length of the cilium. The fibrils are of uniform caliber, and the general arrangement of the fibrils, their number and their sheath show a considerable resemblance to the structure of the spermatozoan tail.

A number of electron micrographs are presented.

REFERENCES

1. Brown, H. P.: On the Structure and Mechanism of the Protozoan Flagellum, *Ohio Journ. Science* 45:247-301, 1945.
2. a. Tremble, G. E.: Some Observations on Nasal Cilia, *Canad. M. J.* 56: 255-9 (Mar.) 1947.
b. Tremble, G. E.: Distribution and Comparison of Nasal Cilia, *A. M. A. Arch. of Oto.*, 1951.

3. Sharpey, W.: On a Peculiar Motion Excited in Fluids by the Surface of Certain Animals, *Edinburgh Med. and Surg. J.* 35:113-122 (July) 1830.
4. Purkinje, J. E., and Valentin, G.: Entdeckung continuirlicher durch Wimperhaare erzeugter Flimmerbewegungen, als eines allgemeinen Phänomens in der Amphibien, Vögel und Säugetiere, *Arch. Anat. Physiol. wiss. Mediz.* 391-400, 1834.
5. Purkinje, E.: Über Flimmerbewegungen im Gehirn, *Müllers Arch. f. Anat. Physiol. u. Wiss.* 1836.
6. Valentin, G.: Flimmerbewegung, *Handwörterbuch d. Physiol.* 1, 1842.
7. Erhard, H.: Studien über Flimmerzellen, *Arch. f. Zellforsch.* 4:309-442, 1910.
8. Gray, J.: Ciliary Movement. *Cambridge Comparative Physiology*, New York, The Macmillan Co., 1928.
9. Gray, J.: The Mechanism of Ciliary Movement, *Proc. Roy. Soc. London* 107:313-332, 1930.
10. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Publishing Co., 1941.
11. Proetz, A. W.: Studies on Nasal Cilia in the Living Mammal, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 42:778, 1933.
12. Proetz, A. W.: Nasal Ciliated Epithelium with Special Reference to Infection and Treatment, *J. Laryng. and Otol.* 49:557, 1934.
13. Hilding, A. C.: The Role of Ciliary Action in Production of Pulmonary Atelectasis, Vacuum in the Paranasal Sinuses and in Otitis Media, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 52:816-833, 1943.
14. Hilding, A. C., and Essex, H. E.: Experimental Production of Negative Pressure in the Frontal Sinus, *Proc. Roy. Soc. Exp. Biol. and Med.* 57:316-318, 1944.
15. Hilding, A. C.: Production of Negative Pressure in the Respiratory Tract by Ciliary Action and its Relation to Post-Operative Atelectasis, *Anesthesiology* 5:225-236 (May) 1944.
16. Freneckner, P.: The Effect of Röntgen and Radium Radiation upon the Action of Cilia, *Acta Oto-Lar.* 27:1 part 297, 2 part 397, 1939.
17. Freneckner, P., and Richtnér, N. G.: Studien über die Zilienbewegungen in den oberen Respirationswegen bei Tieren und Menschen unter normalen und pathologischen Verhältnissen, *Acta Oto-Lar.* 28: 216, 1940.
18. Richtnér, N. G.: Investigations of the Effect of Some New Vasoconstrictors and New Antiseptics upon the Nasal Mucous Membrane of Rabbits, *Acta Oto-Lar. suppl.* 193, 1951.
19. Awarinzew, S.: Beiträge zu der Kenntnis der Flagellaten, *Zool. Anz.* 31: 251, 1907.
20. Bütschli, O.: Braun's Klassen und Ordnungen, *Protozoa* 3, Leipzig, 1889.
21. Prowazek, S. V.: Protozoenstudien. 3 *Euplotes harpa.*, *Arb. a.d. Zool. Inst. d. Univ. Wien.* 14, 1903.
22. Prowazek, S. V.: Flagellatenstudien, *Arch. f. Protistenk.* 2, 1903.
23. Kolzoff, N. K.: Studien ü. Gestalt d. Zelle u. ü d. Spermien d. Dekapoden, *A.f.m.A.* 67, 1906.
24. Jakus, M. A., and Hall, C. E.: Electron Microscope Observations on the Trichocysts and Cilia of *Paramecium*, *Biol. Bull.* 91:141, 1946.
25. Engström, H.: The Structure of Tracheal Cilia, *Acta Oto-Lar.* 39:364-66, 1951.
26. Wykoff, R. W. G.: *Electron Microscopy*, Interscience publ., New York, London, 1949.

27. Weibull, C.: Some Chemical and Physicochemical Properties of the Flagella of *Proteus Vulgaris*, *Biochem. et Biophys. Act.* 2:351-61, 1948.
28. Weibull, C.: Investigation on Bacterial Flagella, *Acta Chem. Scand.* 4: 268-76, 1950.
29. Schmidt, W. J.: Die Doppelbrechung von Karyoplasma etc., *Protoplasma Monographien*, Bonntreager, Berlin, 1937.
30. Ballenger, J. J.: A Study of Ciliary Activity in the Respiratory Tract of Animals, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 58:351-69, 1949.
32. Fischer, A.: Über die Geisseln einiger Flagellaten, *Jahrbuch. f. wiss. Botan.* 26, 1894.
33. Retzius, G.: Biologische Untersuchungen, 1902, et seq.
34. Ballowitz, E.: Zur Lehre von der Struktur der Spermatozoen, *Handwörterbuch d. Naturwiss.* 9, 1913.
36. Bowen, R. H.: Studies on Insect Spermatogenesis, *Jnl. Morph.* 37:79-174, 1922-23.
38. Bretschneider, L. H., and Itersson, W.: An Electronic Microscopical Study of Bull Spermatozoa, *Kan. Ned. Akad. van Wetenschappen* 60, 1947. *ibid* 52:526, 1949.
39. Randall, J. T., and Friedlaender, M. H. G.: The Microstructure of Ram Spermatozoa, *Exp. Cell. Rec.* 1, 1950.

HISTORY OF TRACHEOTOMY

ROBERT E. PRIEST, M.D.

MINNEAPOLIS, MINN.

Tracheotomy was first described just after the beginning of the Christian era. It was credited to Asclepiades who lived in 124 B.C. It probably was first actually performed by Brassavola in 1546. In writing a history of tracheotomy Goodall only found 28 recoveries prior to 1825. Assuming that some obscure reports were not found, and that some cases were unreported, the number is pitifully small for a period of nearly 2000 years. As Goodall says, "It is appalling to think of the vast multitude of unhappy infants who must have fallen victims of laryngeal diphtheria from the Hippocratic era to well on in the 19th century."

The increased prevalence of diphtheria in France in the first quarter of the 19th century was responsible for the final and convincing demonstration of the possibilities of tracheotomy by Bretonneau and Trousseau.

Before the 19th century tracheotomy was contemplated only when suffocation was imminent. An occasional author advocates its early use; de Garengot in 1720 said, "The operation is very dangerous. Few authors say they have performed it, and fewer still that it proved successful. The reason of it seems to be because they perform it too late, and when the patient is dangerously sick."

The word "cyanche," later spelled "syanche" and "squinance," appears throughout early writings on tracheotomy. The term indicates severe inflammatory processes about the pharynx. Quinsy and probably inflammations of the floor of the mouth are included.

In 1546 Brassavola, an Italian physician, described opening the trachea of a boy who was nearly dead from an "abscess in the wind-pipe." This seems to be the first recorded successful case.

In 1620 Habicot reported the case of a 14 year old boy who feared that he was about to be robbed. He wrapped 9 gold coins in

From the Division of Otolaryngology of the University of Minnesota Medical School.

Material presented here was used in course No. 356 at American Academy of Ophthalmology and Otolaryngology in 1947.

cloth and tried to swallow the package. It became impacted in the upper esophagus and pressed so hard on the trachea that it partially suffocated the boy. Habicot opened the trachea. Then with a leaden sound he pushed the parcel into the stomach. In a few days the boy passed money and cloth per anum.

Habicot also reported the case of a boy wounded and left for dead. He opened the boy's trachea and removed a laryngeal blood clot. Habicot had two other successful cases, one an officer with a cut throat, and the other a young woman with a gunshot wound of the throat.

In 1714 Detharding recommended that the operation be done on persons apparently drowned. This recommendation was later reiterated by Heister (1739) and de Pouteau (1783) in order that all water might be got out of the lungs through a tube.

Part of the account of the first British tracheotomy as quoted by Guthrie is given below. The operation was done and reported by George Martine of St. Andrews in 1730.

"I was called to a young lad who was all of a sudden taken ill with a violent trouble in his throat in which however I could see nothing wrong. He had great Pain and Dyspnoea, with an Impossibility of swallowing. I reckoned it an Angina of one of the worst kinds, and the seat of the disease in the larynx and top of the gullet. Notwithstanding repeated Bloodings, Blistering, and Cupping, the Disease continued so obstinate and the patient so like to suffocate that next day in the afternoon, his Friends, although very averse in the morning, when I first proposed the piercing of the Wind pipe, at length earnestly desired that the Operation might be performed; and the poor Lad bade us try any Experiment to preserve his Life. In a few hours he would have strangled to death most miserably. Whence you see it was not out of an itching Desire of making Experiments, or a wanton Officiousness, that we directly set about the Operation. Which was done with such success, that in less than four Days, his Breathing being perfectly easy, we removed the Cannula and left the Glottis to do its own Office.

"Bronchotomy was proposed by Asclepiades 124 B.C., and is described by almost all Writers of Surgery from Paulus of Aegina (625-690 A.D.) and Antyllus 3rd Century A.D.) down to the present time. But they are at so much pains to defend the Reasonableness of it, without mentioning their own Performance of the Operation, that I myself think it has very seldom been reduced to practice.

"Neither Avenzoar nor Albucasis knew any of their Countrymen who had undertaken it. The most I know is that Avenzoar (d. 1162) tried the Experiment on a Goat, which shows the Ingeniousness and Industry of the Author.

"That most accomplished Anatomist and Surgeon, Fabricius ab Aquapendente (1537-1619), frankly acknowledges that he had never ventured to perform it. Neither does his successor, Casserius, pretend to have done it, though he has illustrated the Operation by some very neat Figures.

"The first undoubted and distinctly recorded History I can find of the Operation being actually practised is in the learned Anton. Musa Brasavolus (1500-1570) (com. on Hippocr. de Diet. in acut. iv, 35) who performed it in a des-

perate Squinace¹ and repeated it again in a like case (in 1546). Arnaud, the Frenchman, did it, but his patient died. However, his Countryman Mr. Binard, had better success. (Garengot, 1720, *Operat. Chirurg.*, xxxi, p. 498). Dr. Freind (*Hist. Phys.*, 1725) cites Purmann doing it and tells of another Case by a Surgeon whom he does not name. I hear now that Baxter, a Surgeon in Coupar of Fife, not far from us, and Oliphant in Gask in Pershire, did it with very good Success within these few years."

Here the writer gives a description of the operation. Then he proceeds—

"The Cannula should not be made near so short as is ordinarily proposed. The Parts may be so much tumified that it will require a Pipe above an Inch long to penetrate sufficiently into the Aspera Arteria. There would be less Hazard of a stoppage if the Cannula were shorter and wider. I cannot but think it an ingenious Proposal of one of our Ministers here to make the Pipe double, or one within another; that the Innermost might be safely and easily taken out and cleaned, without any Molestation to the Patient. We found no Inconvenience to our Patient's breathing the Air as it passed through the Pipe, without any cleaning or intercepting Medium, though the House was none of the cleanest, being an ordinary Tradesman's here.

"And now I cannot but notice the needless Pain some Writers are in about healing up the Wound by Bandaging, Stitching, etc. For we found it easily to fill up of itself in a very few days.

"Upon the Subject I should not have had so much to say if this elegant Method of rescuing one from imminent Danger, and the most difficult kind of Death, had not been ordinarily described more from Theory than from Practice, and if Surgeons had been half as bold to assist Nature in such an Extremity as they are officious to disturb her regular and salutary steps."

The technique of tracheotomy evolved slowly. Early operators were afraid to incise cartilage because it was known to heal slowly. They used a transverse incision in skin and trachea.

Tracheotomy tubes were straight and short at first (Fabricius, 1619) and were so illustrated as late as 1795 (Lattas; "Surgery"). Casserius (1561-1616) suggested a curved tube with holes in all directions. Sanctorius (1561-1636) advised a straight trochar such as is used in tapping a hydrocele. Martine was certainly the first to mention a double walled tube but he does not say that he used it.

Purmann of Breslau, in a volume entitled "*Chirurgia Curiosa*" describes "bronchotomy." Purmann is quoted by Guthrie as follows:

"In this manner," he writes, "I opened the Aspera Arteria in Christian Pfennig Mauven, a linen draper at Munden, in 1672. He was 39 years of age, had a violent Swelling in his Throat and was sometimes ready to be choked. The Operation being as happily Performed as could be desired, the Patient was perfectly recovered from Death to Life."

¹i.e., Quinsy.

A most interesting case report is given below. It is quoted from Guthrie's paper and was originally reported in the *Memoirs of the Royal Academy of Surgeons of France*.

"It appears that, in 1733 a London surgeon named Chovell persuaded a condemned man, a highwayman named Gordon, to allow him, for a substantial fee, to perform tracheotomy upon him the night before his execution. This surgeon had tried the experiment on several dogs and always with success, but his human subject, although still alive when cut down by his friends after the hanging, succumbed very soon. That the case aroused considerable interest is shown by the following anecdote, which Louis appends to his account.

"He tells us that shortly after the execution, three London citizens were stopped on the highway by thieves. One of the three, with great presence of mind, passed himself off as Chovell the surgeon. This name rendered the ruffians so polite that they not only returned his purse, but insisted on accompanying him to London, to protect him from any other attack!"

TABLE 1.

TABLE SHOWING DEVELOPMENT OF TRACHEOTOMY
RELATED TO OTHER EVENTS IN HISTORY

(Significant material pertaining to tracheotomy is italicized.)

B.C.

- 7000-2000 Neolithic Age in Europe.
- 2900-2625 Age of Pyramid Builders.
- 580-489 Pythagoras.
- 460-370 Hippocrates.
- 435-432 Meton reforms calendar (365 days).
- 429-347 Plato.
- 124 *Asclepiades of Prusa (Bithynia)—credited by Galen and Aretaeus with being the first to perform tracheotomy.*
- 100 Central heating in Rome.
- 59 Julius Caesar.

A.D.

- 21 Roman baths opened.
- 117-138 *Aretaeus—a great clinician who accurately described pneumonia, diabetes and various kind of insanity, and who never actually performed tracheotomy but credited Asclepiades (see 124 B.C. above) with having done the operation.*
- 131-201 *Galen—was the founder of experimental physiology who supplied theories whenever necessary and left few good clinical case records but did impress his positive views so definitely that European medicine remained at a dead level for nearly 14 centuries. He was the "greatest in experimental demonstration" (Garrison). Credited Asclepiades with tracheotomy.*
- 528 Monte Cassino founded. (Destroyed World War II.)

TABLE 1.—Continued

A.D.	
571-632	Mohammed.
625-690	<i>Paul of Aegina</i> —a great Greek physician who gave original descriptions of lithotomy, tonsillotomy, paracentesis and amputation of the breast. Paul of Aegina quotes a passage from the lost work of Antyllus (who lived in 2nd century, A.D.) <i>suggesting that Antyllus actually did perform tracheotomy and did not merely describe it.</i>
768-814	Charlemagne.
860-932	<i>Rhazes</i> —a great clinician ranking with Hippocrates and Aretaeus as one of the original portrayers of disease. Gave first accurate description of smallpox and measles. <i>Described seeing Ancilisius perform tracheotomy. The original manuscript gave little information about Ancilisius.</i>
1013-1106	<i>Albucasis</i> —born near Cordova in Spain, was author of a great medico-surgical text founded on the works of Paul of Aegina. <i>To prove that survival can occur after the trachea has been injured he sewed up a wound in the neck of a servant girl who had tried to commit suicide by cutting her throat. This case made a great impression on subsequent writers because it is frequently quoted.</i>
1066	Battle of Hastings.
1096-1272	Crusades.
1126-1198	<i>Avenzoar</i> —died at Seville, Spain, was one of the few men who dared to tilt against Galenism. <i>Avenzoar says that when he was a medical student he was anxious to prove that tracheotomy could be done; he therefore successfully performed the operation upon a goat.</i>
1214-94	Roger Bacon.
1223	Cambridge first called a University.
1244-45	Oxford University chartered.
1265-1321	Dante.
1270-1280	Spectacles introduced by Venetian glassmakers.
1330	Introduction of gunpowder in warfare.
1336-1453	Hundred years war.
1348-50	Black death.
1440-1450	Invention of printing.
1452-1519	Leonardo da Vinci.
1490-1553	Rabelais.
1492	Discovery of America.
1497	Vasco da Gama doubles the cape (scurvy on his voyage).
1510-1590	Ambroise Pare.
1514-64	Vesalius.
1517	Sir Thomas More's "Utopia."
1537-1619	<i>Fabricius of Aquapendente</i> , who was Harvey's teacher at Padua, <i>described tracheotomy but had never performed it. He says that in his day it was called "the scandal of surgery."</i>

TABLE 1.—Continued

A.D.	
1546	<i>Antonius Musa Brassavola</i> , an Italian physician, <i>described opening the windpipe of a boy who was nearly dead from an "abscess in the windpipe."</i> This appears to be the first recorded successful case.
1558-1603	Reign of Queen Elizabeth.
1561	Fallopian published "Observation Anatomicae" (Fallopian tubes). Pare founded orthopedics.
1651-1626	Francis Bacon.
1564-1616	Shakespeare.
1564-1642	Galileo.
1565	Jean Nicot brings tobacco plant to France.
1578-1657	William Harvey.
1590	Compound microscope invented by Hans and Zacharias Janssen.
1595	Mahuang (<i>Ephedra</i>) noted by Shi Cheng Li.
1600	Queen Elizabeth charters the East India Company.
1602	Hamlet produced. Harvey graduates (M.D.) at Padua.
1604	Johan Kepler demonstrates inversion of optic image on retina.
1606-69	Rembrandt.
1609	Henrik Hudson anchors "Half Moon" in New York Bay.
1620	Galileo invents telescope. Landing of Pilgrims in America.
1620	<i>Nicolas Habicot of Paris reported four successful tracheotomies published the first book on tracheotomy (108 pages).</i>
1639	First printing press in North America.
1641	<i>Fontanus of Amsterdam—one successful tracheotomy in a woman.</i>
1642-1727	Sir Isaac Newton.
1646	<i>Rene Moreau, Paris—two tracheotomies in male adults.</i>
1660	Schneider upsets Galen's theory that nasal secretion originates in the pituitary gland.
1663	First hospital in American colonies, New York.
1665	Newton discovers binomial theorem and law of gravitation.
1666	Great fire of London. Newton described solar spectrum. Coroners appointed for each county in Maryland.
1671	<i>Meyssonier of Lyon—three tracheotomies of which neither sex or age are stated.</i>
1672	<i>Godfrey Purmann, of Breslau—one tracheotomy in a man aged 39.</i>
1689	<i>Smalsius, of Leyden—one tracheotomy in a man.</i>
1694	<i>Fulvio Gberli—one tracheotomy in a man.</i>
1711	John Shore invented the tuning fork.
1718	<i>Heister, of Helmstadt—one tracheotomy in a man.</i>
1724-1804	Kant.

TABLE 1.—Continued

A.D.	
1730	<i>George Martin, at St. Andrews—one tracheotomy in a boy; this appears to have been the first successful British case; mentions the double walled tube.</i>
1740	University of Pennsylvania founded as "College of Philadelphia."
1745	Barbers separated from higher surgeons in England (Act of June 24).
1743	<i>Virgili, of Cadiz—one tracheotomy in a soldier aged 23.</i>
1748	<i>Bauchot, of Port Louis—two tracheotomies; one in a man aged 23 and one in a woman aged 72.</i>
1765	<i>A French surgeon—one tracheotomy in a woman of 35.</i>
1766	<i>Caron—one tracheotomy in a boy aged 7 years for removal of a bean.</i>
1779	<i>Ferriere—one tracheotomy in a man aged 50 years.</i>
1782	<i>Andrie, of London—one tracheotomy in a boy of 5 years.</i>
1792	<i>John Hunter, of London—one tracheotomy in a man.</i>
1802	<i>Flajani, of Rome—one tracheotomy in a young man.</i>
1814	<i>Chevalier, of London—one tracheotomy in a boy aged 7 years.</i>
1815	<i>William Lawrence—one tracheotomy in a man aged 53.</i>
1771-1862	Brettoneau } actually used tracheotomy in the modern sense, mainly
1801-1867	Trousseau } in diphtheria.

REFERENCES

1. Guthrie, Douglas: Early Record of Tracheotomy, *Bull. Hist. Med.* 15:59-64 (Jan.) 1944.
2. Goodall, E. W.: The Story of Tracheotomy, *Brit. J. Child Dis.* 31:167-176 and 252-272, 1934.
3. Wright, Johnathan: The Nose and Throat in Medical History, publisher and year not given but copy of book available in Hennepin County Medical Library, Minneapolis.
4. Leonardo, Richard A.: *History of Surgery*, Froben Press, New York, 1943.
5. Garrison, Fielding H.: *An Introduction to the History of Medicine*, Fourth Edition, W. B. Saunders Co., Philadelphia, 1929.

LXXXII

A PROLONGED LOCAL ANAESTHETIC IN CONTROL
OF POST-TONSILLECTOMY PAIN

MORRIS DAVIDSON, M.D.*

ST. LOUIS, MO.

ROBERT G. BOLES, M.D.

LOUISVILLE, KY.

AND

SANFORD C. SNYDERMAN, M.D.

FORT WAYNE, IND.

A solution of procaine and butyl aminobenzoate in a stable water-miscible, non-oily vehicle (Efocaine) has been recommended for the control of post-operative pain.

Iason and Shaftel¹ have reported excellent results with the use of this agent in rectal and abdominal surgery.

Penn² has reported good results with this solution in the control of post-tonsillectomy pain.

In the present report tonsillectomies were performed on eight children and nineteen adults to determine the effect of this agent in controlling post-tonsillectomy pain.

General anesthesia was used on the eight children, and the tonsils were removed by the dissection and snare technique. When the fossae were entirely dry 1.5 cc of the solution was injected submucosally at several points along both tonsil pillars on the left side only. Care was taken not to deposit the solution too superficially.

Results with eight children so treated were:

- 2 reported no pain on either side of the throat,
- 3 complained of equal pain on both sides of the throat,
- 1 stated the injected side felt better,
- 2 thought the pain on the injected side was worse.

*From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri.

Because it was felt that the responses from the children were not entirely reliable, the anesthetic was used in nineteen adults. All the adults were males between the ages of 18 and 30 years. Anesthesia for the tonsillectomies in the adults consisted of local procaine, and the tonsils were removed by the dissection and snare method. When the fossae were entirely dry, 1.5 cc of the solution was injected along the anterior and posterior pillars of the right side only. Here again, care was taken to inject submucosally, and superficial infiltration was avoided. None of the patients were aware that they were participating in this special study. They were checked daily and remained in the hospital on an average of eight days.

Results with nineteen adults so treated were as follows:

- 4 patients stated that the injected side felt better,
- 5 complained of equal pain on both sides of the throat,
- 10 felt that the injected side hurt worse than the opposite side.

SUMMARY

27 patients subjected to tonsillectomy were treated with a solution of procaine and butyl aminobenzoate to evaluate its effect in reducing post-tonsillectomy pain.

It is felt that this agent is of questionable value in reducing pain following tonsillectomy.

35 N. CENTRAL AVE.

703 BROWN BLDG.

629 WAYNE PHARMACAL BLDG.

REFERENCES

1. Iason, A. H., and Shaftel, H. E.: A New Approach to the Problem of Post-operative Pain, *Am. J. Surg.* 83:549-555, 1952.
2. Penn, S. E.: Control of Post-tonsillectomy Pain, *Arch. Oto.* 56:59-60, 1952.

LXXXIII

CYSTS OF THE THYROGLOSSAL DUCT AS A COMPLICATING FACTOR IN LARYNGEAL OPERATIONS

FREDERICK A. FIGI, M.D.

AND

HUGH A. JOHNSON, M.D.

ROCHESTER, MINN.

The coexistence of a carcinoma of the larynx and a persistent cyst of the thyroglossal duct in the same patient is a rare occurrence. The former condition is essentially a disease of the degenerative period of life while the latter generally is encountered in childhood. Sixty per cent of these congenital anomalies are discovered in patients less than 10 years of age. Two cases of the combined lesion were encountered recently in the Section of Plastic Surgery and Laryngology at the Mayo Clinic. A review of the medical literature of the past twenty years has failed to reveal any reports of similar cases.

The presence of the two lesions in the same patient is important in considering the treatment of the more lethal one. First, the cyst of the thyroglossal duct may suggest extralaryngeal extension of the tumor and may lead the surgeon to plan a more radical operation than necessary, or, because of the apparent extrinsic involvement, radiation therapy may be advised. Second, the treatment of a cyst of the thyroglossal duct is surgical removal of the entire lesion together with a section of the hyoid bone.¹⁻³ This is a more time-consuming procedure than thyrotomy alone. Failure to remove the cyst completely and to resect the midportion of the hyoid bone is likely to result in a recurrence of the cyst.⁴

A persistent thyroglossal duct results from an embryologic anomaly in the development of the thyroid gland.⁵⁻⁷ The thyroid anlage is an outpouching of the floor of the primitive pharynx. The distal end of this pouch becomes the thyroid gland and the connecting neck, if it persists, the thyroglossal duct. This is usually, but not always, situated in the midline.¹ The duct normally atrophies during the sixth week, but its point of origin on the tongue is permanently indicated by the foramen caecum. The distal attachment of the duct may persist as a pyramidal lobe of the thyroid.⁸ In the

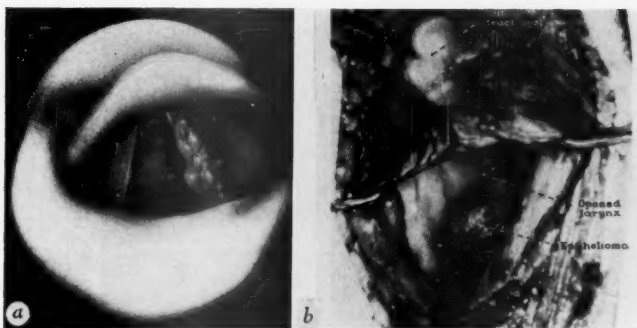


Fig. 1.—*a*, Epithelioma of left vocal cord; *b*, appearance of cyst of the thyroglossal duct and the epithelioma of the left vocal cord after larynx was opened.

course of development, the pouch passes through the anlage of the hyoid bone, which explains the occasional attachment of a pyramidal lobe to that structure. All stages of the development, including the fistula, the sinus (infected or noninfected), the cyst, on down to the duct or merely the epithelial cord at the site of origin, have been observed in adults.

REPORT OF CASES

We shall report briefly 2 cases in which a cyst of the thyroglossal duct was encountered in the course of an operation which was performed primarily for an epithelioma of the larynx.

CASE 1.—A man, aged 47 years, came to the clinic on March 16, 1949, because of hoarseness which had been present for four months. Laryngeal examination disclosed a tumor which was situated in the middle of the left true vocal cord and involved two thirds of this structure (Fig. 1*a*). Biopsy revealed that the tumor was a squamous cell epithelioma, grade 2. Physical examination and laboratory tests did not reveal any other abnormality.

Operation was performed on March 19, 1949. An incision which was carried down to the thyroid cartilage disclosed a cystic tumor which was situated in the space between the hyoid bone and the thyroid cartilage. After the cystic tumor had been dissected backward for a short distance, it appeared to be a cyst of the thyroglossal duct (Fig. 1*b*).

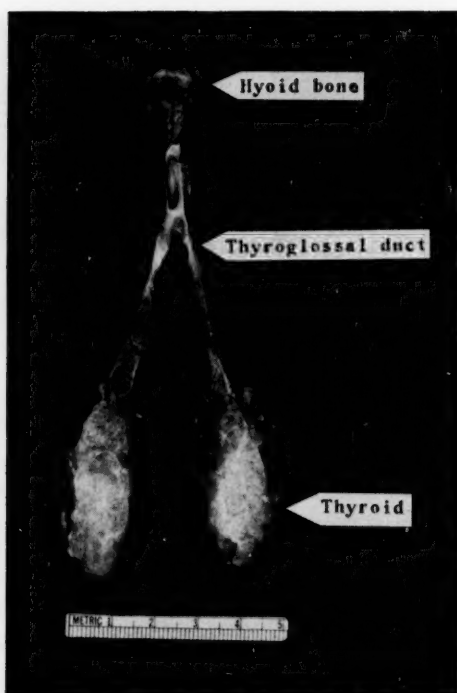


Fig. 2.—Excised specimen showing segment of hyoid bone, persistent thyroglossal duct and attached adenoma of the thyroid isthmus.

At this stage of the operation, the larynx was opened. The epithelioma of the left vocal cord was excised widely and the resulting wound was electrocoagulated thoroughly.

The cyst of the thyroglossal duct then was dissected backward almost to the base of the tongue where it gradually tapered to a point. Since the tract was attached to the hyoid bone, it was necessary to remove a segment of this bone in order to completely excise the tract. The transverse diameter of the cyst was 2 cm and its longitudinal diameter was slightly longer. The cyst was lined with epithelium. The pathologist who examined the surgical specimen said that the cyst had been removed completely. A temporary tracheostomy was then performed.

When the patient returned to the clinic for a checkup on July 29, 1949, examination did not disclose any evidence of a recurrence

of the cyst or the epithelioma. The patient died of coronary disease on October 6, 1951. Although necropsy was not performed, there was no clinical evidence of a recurrence of the carcinoma or of the cyst of the thyroglossal duct.

CASE 2.—A man, aged 58 years, came to the clinic on February 5, 1951, because of hoarseness which had been present for six weeks. Laryngeal examination revealed a thickening of the left true vocal cord, more marked anteriorly. Biopsy revealed that the thickening was a squamous cell epithelioma, low grade 2. Physical examination and laboratory tests did not reveal any other abnormality.

Operation was performed on February 9, 1951. When the midline incision was made to expose the larynx, a cystic tumor was observed in the notch of the thyroid cartilage. An extension of the cyst was traced caudad to an adenoma of the isthmus of the thyroid gland. Since the adenoma was lying directly over the trachea, it was necessary to excise it along with the cyst to form a temporary tracheal stoma (Fig. 2). The cyst was then dissected cephalad, by excising the midportion of the hyoid bone through which the tract passed. The tract ended just beyond the hyoid bone. The cyst and tract measured about 1.5 by 3 cm.

The larynx was then opened. The tumor of the left true vocal cord was excised widely and the resulting wound was electrocoagulated thoroughly.

On examination, November 29, 1952, there was no evidence of recurrence of either lesion.

SUMMARY

Persistent thyroglossal duct cyst is a rare complication of laryngeal operations but is important because it might suggest extralaryngeal extension of a malignant tumor of the larynx, resulting in poor judgment in the treatment. Secondly, proper treatment of a persistent cyst of the thyroglossal duct prolongs laryngeal operations considerably.

MAYO CLINIC.

REFERENCES

1. Ward, G. E., Hendrick, J. W., and Chambers, R. G.: Thyroglossal Tract Abnormalities—Cyst and Fistulas: Report of 105 Cases From the Johns Hopkins Hospital Observed During the Years 1926 to 1946, *Surg., Gynec. and Obst.* 89: 727-734 (Dec.) 1949.
2. Sistrunk, W. E.: Surgical Treatment of Cysts of Thyroglossal Tract, *Ann. Surg.* 71:121 (Feb.) 1920.

3. Sistrunk, W. E.: Technique of Removal of Cysts and Sinuses of the Thyroglossal Duct, *Surg., Gynec. and Obst.* 46:109-112 (Jan.) 1928.
4. Marshall, S. F.: Thyroglossal Cysts, *Lahey Clin. Bull.* 6:2-6 (July) 1948.
5. Weller, G. L., Jr.: Development of the Thyroid, Parathyroid, and Thymus Glands in Man, *Contrib. Embryol.* 24:93-139, 1933.
6. Norris, E. H.: The Early Morphogenesis of the Human Thyroid Gland, *Am. J. Anat.* 24:443-465 (Nov.) 1918.
7. Arey, L. B.: *Developmental Anatomy: A Textbook and Laboratory Manual of Embryology*, Ed. 5, Philadelphia, W. B. Saunders Company, 616 pp., 1946.
8. Callander, C. L.: *Surgical Anatomy*, Ed. 2, Philadelphia, W. B. Saunders Company, 858 pp., 1939.

THE TREATMENT OF DEAFNESS BY PROSTHESIS

H. G. KOBRAK, M.D.

CHICAGO, ILL.

In certain sound conduction lesions, there is a destruction of the ear drum and parts of the ossicular chain. It has been tried to replace the destroyed eardrum and the missing ossicles by materials brought into the tympanic cavity. These materials are known as hearing prostheses.

The literature on hearing prostheses is very large and dates back more than three centuries.

In 1640 Marcus Banzer published a paper on hearing defects and the treatment of deafness. In the paper, Banzer advises the use of a small tube of elk's claw covered by a pig's bladder at its end. A century later in 1763 Leschewin reports on the use of an artificial eardrum and in 1815 Authenrieth proposed his artificial eardrum for the improvement of hearing. In spite of a series of favorable reports by Ward, Delstanche and Tröltzsch the method was not adapted generally until Toynbee in 1852 introduced the artificial eardrum, which for a long time carried his name. A wave of enthusiasm followed Toynbee's publication because lay people expected from it the same therapeutic possibilities as glasses provide for the eye. Needless to say that the expectations were not fulfilled. In certain cases there was a striking improvement of hearing. However, the disadvantages and restrictions of the method soon became apparent. The prosthesis irritated the delicate tissues of the middle ear. Secretion recurred or increased in amount. Noises due to mastication, talking or due to spontaneous shifting of the instrument were bothersome.

Ever since the original conception of the method, physicians have attempted to overcome the difficulties. The ideal solution is a combination of maximal hearing improvement with minimum irritation. The European literature of the second half of the 19th century and the early nineteen hundreds is full of descriptions of the mechanical prostheses in the ear. In this country serious and inspired

From the Division of Otolaryngology, The University of Chicago.

The experiments cited in this paper were supported in part by the Veterans Administration Contract V1001M-2846.

attempts were made by the Doctors G. and M. Pohlman during the last fifteen years. Again as in Toynbee's days a wave of interest and enthusiasm has been sweeping the country finding its climax in a publication in a popular magazine. It seems that history repeats itself. Too great promises are bound to be followed by disappointment, disappointment among both the layity and the medical profession. An objective detached appraisal seems in order.

The otologist should be interested in the prosthesis because it represents a simple but rather effective method to improve the hearing of the patient. The physiologist is bound to gain new information about the function of hearing. Every patient treated with an acoustic prosthesis represents an experiment on the physiology of hearing.

A re-study of the problem of the hearing prosthesis seems to be in order for the following reasons:

1. Older publications expressed the hearing gain only in terms of whispered voice and tuning fork tests without proper attention to masking. Therefore, controlled audiometric tests seemed necessary.
2. Modern materials, like plastics and biologically non-irritating metals like Tantalum are available today which were not accessible to previous experimentators.
3. The availability of antibiotics makes procedures possible which were too dangerous a few decades ago because of the danger of infection.
4. Our knowledge of the physiology of hearing is more advanced today. The placing of the prosthesis in various parts of the middle ear is based on more exact knowledge than previously.
5. The method of direct cinematographic recordings of the ear which was developed during the last few years at the University of Chicago promised to give a new approach to the study of the ear prosthesis.

A multitude of mechanical devices have been used as prostheses. Three main groups can be differentiated.

1. Devices which close a perforation of the eardrum. (Eardrum prosthesis)
2. Devices which are attached to certain parts of the medial tympanic wall, mostly in the round window or stapes area (tympanic prosthesis).
3. Prostheses which aim to replace a destroyed ossicular chain (ossicular prosthesis).

THE EARDRUM PROSTHESIS

Physiological Considerations: A patient who has a normal ossicular chain but a central perforation presents the following four biophysical abnormalities of sound conduction in the ear.

1. Sound energy can flow unimpeded into the middle ear. Both sides of the drum membrane are exposed to the same phase of the sound wave.

2. Sound energy can reach both windows directly. The preferential sound conduction through the ossicular chain to the oval window is abolished.

3. The difference in area between eardrum and stapes footplate (the so-called hydraulic ratio) is diminished.

4. The air of the middle ear is normally encased behind the eardrum. An opening in the eardrum represents a change from an enclosed-air bubble to an open air space.

The closure of the perforation reverses these four pathological conditions:

- a. The tympanic membrane is again only exposed to sound on one side.

- b. The preferential sound conduction by way of its ossicles is re-established.

- c. The hydraulic ratio is restored.

- d. The air bubble in the middle ear becomes encased again.

At the present time it is not possible to ascertain the audiometric importance of each of the four factors. The main difficulty for an analysis of these factors appears to lie in the individual differences which the various ears show. The variations of eardrum thickness, relative positions of the two inner ear windows, the size and shape of the middle ear spaces and the physiological variations in the impedance of the eardrum are still confusing the student of sound conduction. Therefore the audiometric hearing loss due to a given eardrum perforation cannot be analysed in each case. Clinical observations show great variations of both the frequency range and the amount of hearing loss. A number of our patients were observed under low power magnification (eardrum microscope). The impression was obtained, that those patients whose pathology consisted only of the eardrum perforation but who had an essentially normal ossicular chain had always a good hearing gain when an eardrum prosthesis was applied. The majority of the audiograms showed

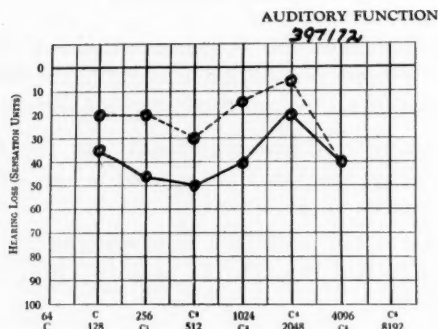


Fig. 1.—Audiogram of a patient with central perforation. The solid line indicates unaided hearing by air conduction. The broken line designates hearing threshold obtained by closing the perforation with a cotton pellet.

a hearing gain of the low frequencies with no hearing improvement of the higher tones. The amount of hearing gain was considerable. Twenty-five to 35 db were frequently observed. The restoration of the hydraulic ratio seems to be the most essential factor of the treatment.

THE EARDRUM PROTHESIS

CLINICAL CONSIDERATIONS

For more than 50 years attempts to close an eardrum perforation by application of Trichloracetic acid have been described in various intervals in the literature (Okuneff, Dunlap, Dunlap and Schuknecht, Clark). All authors agree that many perforations can be closed permanently. Unfortunately the method is a rather tedious and time consuming but gains outweigh by far the disadvantages of the method. The permanent closing of the perforation restores the patient back to ear-normalcy. His hearing is improved and his middle ear is protected against water entering from the outer ear canal. The natural closure of the eardrum perforation is to be preferred to all artificial devices by which an eardrum can be closed. However, many patients cannot make as many office calls as necessary for this type of treatment. Furthermore a certain type of eardrum perforations is not suitable for closure. Some perforations fail to heal without any apparent reason. For this group of patients the prosthesis offers the advantage of improved hearing (Fig. 1). The best cases are bilateral middle ear disease with bilateral hearing impairment. Patients with unilateral hearing impairment do not ex-

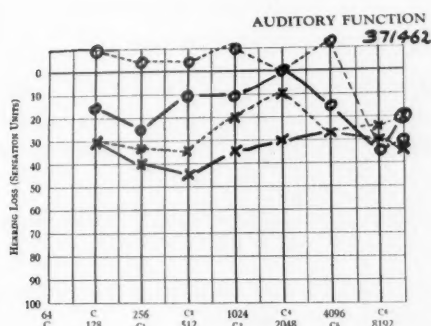


Fig. 2.—Audiogram of patient with bilateral large perforation of eardrum. Hearing improvement was obtained by closing perforation with latex rubber membrane. Solid lines indicate hearing without prosthesis. Dotted lines designate threshold of hearing with prosthesis.

NOTE: Sufficient hearing gain is mostly obtained by unilateral usage of an eardrum prosthesis. This patient insistent on bilateral fitting.

perience the spectacular and gratifying sudden hearing gain when the prosthesis is applied. Directional hearing, however, is improved in these patients. The case of a business executive who had to attend important board meetings is apropos. This patient was handicapped in understanding the discussions going on both on his right and left side. On his left side he had a hearing loss of 45 db. The prosthesis raised his hearing on the left side to about 15 db. He could now follow the discussion on each side without difficulty. A woman patient who had to drive many hours, was handicapped by her right-sided hearing defect. She could, therefore, not converse with a rider sitting on her right side. A prosthesis solved her problem.

Bilateral use of the prosthesis is rare, but some patients insist on wearing a prosthesis in each ear (Fig. 2).

Several materials have been suggested in the literature. A pellet of cotton, soaked in mineral oil, Cargyle membrane, cellophane, cigarette paper, latex rubber, wool and many others. It is not apparent that any of these materials is superior in its acoustical effect. However, the amount of irritation differs. The oil soaked cotton is irritating when used for longer periods and is not advisable. Membranes can be placed by the otologist and left in situ for many weeks. Regular bimonthly supervision by the otologist is advised. Derlacki has advised combining the closure of the perforation with the wearing of a prosthesis during the treatment period.

THE TYMPANAL PROSTHESIS
PHYSIOLOGICAL CONSIDERATIONS

The loss of eardrum and of the two lateral ossicles as found in advanced chronic middle ear infections and in a radical mastoid cavity represents the following abnormal conditions of sound conduction:

1. The normal impedance matching accomplished by the eardrum and the ossicular chain is no longer present.
2. The preferential sound conduction to one inner ear window by the ossicular chain is abolished.
3. The two windows face the sound source and execute vibrations with nearly equal phase instead of opposite phase as in normal sound conduction.
4. The round window membrane does not face an encased air bubble but an open air space. The vibratory phenomena in the cochlea especially the dampening may be influenced by this condition.
5. The sound conduction system represents a mass which is in direct contact with the cochlea. The loss of the eardrum, malleus and incus diminish this mass.
6. The effect of the tensor tympani muscle is lost.
7. The effect of the stapedius muscle is changed. The stiffness of the stapes alone is smaller than stiffness of the intact ossicular chain. Visible motions of the stapes are observed during contraction, which do not occur on the intact chain.

At the present time, it is impossible to assess these physical factors in diseased ears as to their individual role in causing the hearing loss. The attempt was made to obtain new information by utilizing stroboscopic cinematographic methods.

DIRECT OBSERVATIONS OF THE ACOUSTIC VIBRATIONS
OF EAR PROTHESIS

The sound conduction system is an extremely complicated physical system. Clinical observations are unsatisfactory for its study because each pathological case has individual features which make comparisons and generalizations impossible. The attempt has been made to overcome some of the difficulties by studying the function

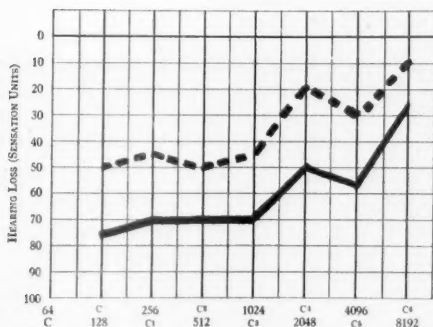


Fig. 3.—Audiogram of a patient with radical mastoid cavity. The unaided hearing of this patient (solid line) is insufficient for ordinary working conditions. A tympanal prosthesis raises the threshold (broken line) to such an extent that an electrical hearing aid can be avoided.

of the middle ear under controlled laboratory conditions. The method which has been described before¹³ consists in utilizing fresh temporal bone specimens of deceased patients. These ear specimens can be stimulated by acoustic signals. The oscillations can be photographed and measured on the screen for quantitative analysis. The effect and the mechanism of the prosthetic devices was thus studied on fresh temporal bones. The observations can be summarized as follows:

Some of the substances commonly used as prosthesis material (cotton pellets, plastics) do not show acoustic vibrations as readily as a normal ossicular chain. However, if these materials are used in moist ears or introduced with moisture (e.g. water or mineral oil) acoustic vibrations of the fluid can be seen. The fluid contents of the prosthesis can be either a film of moisture over a plastic tube or a drop encased in the fiber-pattern of a cotton pellet. When observing the film recordings, one obtains the impression that the cotton pellet with its fiber system is the skeleton which by its adhesion keeps the vibrating fluid in its place and in its shape. Similar observations can be made on the Koragel insert. The fluid layer on the plastic shows definite vibrations while the tube itself does not oscillate.

It can be assumed therefore that the tympanal prosthesis consists of two distinct parts:

1. The moisture which is the acoustically active ingredient (oil, saline or purulent secretion);

2. The skeleton which holds the drop of moisture in place (cotton pellet, plastic tube, tantalum mesh, etc.)

Clinical observations show that a plastic tube inserted in a dry ear does not improve hearing. It has been recommended for wet ears only. Similarly dry cotton pellets are not effective. However, if the cotton pellet is dipped in oil or saline and reinserted, a hearing improvement may be noted immediately.

THE OINTMENT PROSTHESIS

Realizing the duplicity of the tympanal prosthesis the attempt was made to utilize a material which contains the liquid and provides fixation of position simultaneously.

Aquaphor ointment was chosen because it has the ability to hold three times its own weight of water. The water binding ingredient in aquaphor is a group of esters of cholesterol isolated from wool fat. It adheres to skin and to the moist mucous membrane. It keeps indefinitely and is odorless. The main advantages is that it does not irritate the middle ear. We have placed the ointment into the tympanic cavity and left it for weeks and months without observing undue reactions. Aquaphor can be sterilized. In every case it was applied under sterile precautions.

Aquaphor is recommended especially in cases in which the niche of the round window is visible and accessible. The plugging of the round window niche by aquaphor offers an acoustic effect which can be maintained for weeks or months without irritation.

Prerequisite for a successful application of aquaphor is however that the patient is suitable for the fitting of an acoustic prosthesis. Aquaphor cannot produce a hearing gain, where other types of hearing prosthesis fail. The value of the ointment prosthesis is based on two factors:

1. The absence of irritation.

2. The possibility of reactivation of the prosthesis after the acoustic effect fades out. A drop of fluid can be inserted into the middle ear by the patient (sterile saline at body temperature). The fluid will be absorbed by the ointment. Return of the hearing improvement is observed by this self treatment.



Fig. 4.—The mechanism of the tympanal prosthesis. An enlarged view of the human cochlea and the two fenestrae. The stapes (S) is seen in the niche of the oval window. The tendon of the stapedius muscle emerges from the pyramidal process and attaches on the posterior aspect of the head of the stapes. The niche of the round window (R) is located below the oval window. The distance is about 4 mm. The cochlea of this specimen has been opened and both scalae of the basal coil are visible. The scala vestibuli is in open communication with the vestibule (V) while the scala tympani (t) ends at the round window membrane (m). The cochlear partition executes a 90 degree turn (arrow) near its basal end. A part of the basilar membrane is visible near the round window.

The normal stimulation of the cochlea consists in a vibration of the cochlear partition. When the stapes pushes inward, the cochlear partition bulges into the scala tympani and consequently the round window membrane bulges outward. During the outward motion of the stapes, the corresponding movements are opposite.

In certain sound conduction lesions, there is no preferential sound conduction to the oval window by way of the ossicular chain. Both windows are exposed directly and equally to the sound.

The purpose of the tympanic prosthesis is to re-establish inequality of sound conduction to the two windows. This is accomplished either by blocking of the round window or by changing the phase of the sound. The exact mechanism by which the tympanal prosthesis works is not clearly understood. The position of the prosthesis is indicated by the double line. In placing the prosthesis it is essential that a small air bubble remains between the round window membrane and the prosthesis.

CLINICAL CONSIDERATIONS

The following practical procedure is recommended. When a patient has a considerable loss of hearing due to a destructive middle ear lesion, the possibilities of a hearing prosthesis should be considered. First, it should be elucidated from the history whether or not the patient has spontaneous improvement of hearing. Fairly frequently, a drop of secretion acts as spontaneous prosthesis and produces fleeting hearing improvement. Attention should be paid by the otologist whether or not yawning has the tendency to improve hearing even for only a short time.

The most frequent site of the prosthesis is the niche of the round window. Therefore it may be wise to begin the examination by placing a moist cotton pellet into the round window area.

The size of the pellet may be of importance, equally important is the exact position. The patient should be exposed to a constant sound. One can play the radio, let the water run or one may keep talking continuously into the ear of the patient. The pellet is then moved around in the tympanic cavity with an ear hook. The exact positioning is very critical. No analytical rule can be given as to the positioning of the prosthesis. It is, unfortunately a procedure of trial and error. It is impossible to predict from the otoscopic appearance even by using ear microscopic examination whether or not an ear is suitable for prosthesis fitting and in which part of the ear the prosthesis should be placed. About half of our patients tested receive benefit from a prosthesis.

Some patients have a hearing loss which is so severe that an electronic hearing aid would be indicated. The hearing gain obtained by a tympanal prosthesis is in some cases so pronounced that the patient can do without the electric hearing aid. Figure 3 is a typical case. The tympanal prosthesis in such a specific case is in our opinion superior to an electronic hearing aid. It provides an improvement of hearing of 35-40 db, it is invisible, very small, extremely simple and negligible in cost. Many patients learn to insert the cotton pellet prosthesis or a plastic tube into the tympanic cavity. Although these patients are completely independent from the otologist regular visits to the otologists are recommended. These patients may be fitted by the otologist but learn to re-activate the effect by installation of fluid into the tympanic cavity.

All procedures done in the tympanic cavity should be carried out under aseptic precautions.

THE OSSICULAR PROSTHESIS

Ever since Marcus Bauzer's original publication in 1640 attempts have been made to place into the middle ear cavity materials which replace the eardrum and the ossicular chain. Augustus Pohlman devised a prosthetic appliance which was based on the physiological effect of the columella in the bird's ear. Present physiological concepts attribute to the avian tympanic structures a function which is essentially the same as the mammalian ossicles: namely, an impedance matching function. The most important feature in the avian and mammalian middle ear is the difference in size between the eardrum and the stapes. Pohlman constructed a rubber membrane held and kept under tension by a silver ring in the depth of the outer ear canal. A nylon rod was placed through a central perforation of the membrane and brought in contact with the stapes or any other functionally active point in the otic capsule (round window, surgical fenestra in lateral semicircular canal). The theoretical basis of the Pohlman prosthesis is absolutely sound, and clinical evidence has been accumulated that it produces gratifying hearing improvements.

Unfortunately, however, there are many technical steps involved in the fitting of the ossicular prosthesis which make the procedure rather cumbersome. Many otologists therefore who began work with the Pohlman prosthesis have discontinued the method because of the difficulties in making and fitting the prosthesis. Furthermore, the maintaining of the hearing gain proved to be difficult because of the loose contact between nylon rod and head of stapes. The hearing improvement was lost frequently when the nylon rod "fell off" the stapes. Pohlman's original advice was to use the ossicular prosthesis in dry ears, e.g., a dry radical mastoid cavity or a dry tympanic cavity with large eardrum and ossicular defects. For secreting ears he advised a plastic tube, which is much easier to fit, but satisfactory in regard to the hearing gain. It must be agreed that a dry ear and a moist ear pose different prosthetic problems, because the moisture is an acoustically important part of the prosthesis. We believe that the solution to the fitting problem of the dry ear is to "import" the moisture into the tympanic cavity. The Aquaphor salve contains the moisture and acts together with the ointment base which serves as anchor. The Aquaphor is of course not the only salve which may be suitable. Other ointments may possess similar moisture holding capacities and may be useful as tympanic prosthesis. The salient point in this discussion is that the clinical differentiation between dry and wet ear for the selection of a prosthesis is no longer essential.

Therefore the construction of an ossicular prosthesis is not a necessity for dry ears.

SUMMARY

1. In certain sound conduction lesions, foreign material may be placed into the middle ear with the purpose of replacing the destroyed eardrum and ossicles. These materials are known as prostheses.

2. Three types of prostheses can be distinguished.

a. The eardrum prosthesis, which covers a perforation of the eardrum.

b. The tympanal prosthesis which is placed in the niche of the round window, the niche of the oval window or against a surgical fenestra.

c. The ossicular prosthesis which replaces the destroyed eardrum and ossicular chain in form of a rubber membrane with a rod pushed through the center of the membrane. Contact is made with the stapes, round window, a temporal fenestra or any other "trigger point."

3. The eardrum prosthesis is useful in cases in which the usual therapy of natural closure cannot be carried out or during the treatment of closing.

4. A favorable material for an eardrum prosthesis is latex rubber, other materials are suitable also. Aseptic precautions are recommended in positioning the prosthesis. The hearing gain obtained varies, but a 30 db. hearing improvement for some frequencies is frequently found.

5. The mechanical principle by which the eardrum prosthesis acts is most likely the restoration of the hydraulic ratio of the sound conduction system. Further biophysical experiments are needed to clarify the effect.

6. The tympanal prosthesis is useful in ears with extensive destruction of eardrum and ossicular chain especially valuable in a radical mastoid cavity. The material frequently used consists in a cotton pellet soaked in oil.

7. The mechanism by which the tympanal prosthesis acts is not clearly understood.

8. Experiments on cadaver ears revealed that a tympanal prosthesis consists of two essential parts.

a. Moisture which is the acoustically important ingredient.

b. A skeleton of fibers (e.g. cotton) or a plastic tube which maintains the position and the shape of the moisture.

Direct observations on the acoustic vibrations of the tympanic prosthesis were made by stroboscopic cinematographic recordings.

9. The placing of foreign material into the tympanic cavity should be done under minimal mechanical irritation. Aquaphor ointment was found to be the least irritating material. It consists of an ointment base and moisture absorbed and carried in the salve. It is useful as a tympanic prosthesis. Patients can "reactivate" their prosthesis by installing a drop of fluid into the middle ear cavity (normal saline at body temperature).

10. The Aquaphor ointment is essentially recommended for patients in whom the niche of the round window is accessible. A small amount of ointment can be inserted into the ear with an earhook and be used to plug the round window niche. It can be left in the round window niche indefinitely. The ointment can be sterilized and should be inserted under aseptic precautions.

11. The ossicular prosthesis (diaphragm-rod prosthesis of Pohlman) is an ingenious device which is constructed like the columella of a bird's ear. It is physiologically well founded, and provides good hearing improvements in some cases. However technical difficulties in making, fitting and maintaining the diaphragm rod prosthesis are so great, that it has not found general acceptance.

12. A mechanical prosthesis should be tried in every suitable patient. The prosthesis is simple, inexpensive, invisible and effective. Proper materials, especially the Aquaphor ointment can be left in the ear for weeks or months without causing irritation or reactions. Regular supervision by the otologist is advised.

REFERENCES

1. Autenrieth and Bohneberger: *Tübinger Blätter f. Naturwissenschaften und Arznei Kunde*, Vol. 1, 1815.
2. Aubry, M.: *Ann. d'Otolaryngol.* 64:167, 1947.
3. Asherson, N.: *Journal of Laryngology and Otology*, 64:239, 1950.
4. Banzler, Martin: *Disputatio de auditione laesa*, 1640.
5. Delstanche, C.: *La Clinique* 29, 30, 31, 1887. *Ref. Arch. Ohren. Nasen u. Kehlkopfheilkd.* 26:252.
6. Clark, A. F.: *Texas State T. M.* 46:145, 1950.
7. Derlacki: *Lecture before Chicago Laryngological and Otological Society*, Jan. 7, 1952.
8. Dunlap, A. M.: *Repair of the Tympanic Membrane in Perforations of Long Standing*, *Laryngoscope* 27:81-85, 1917.

9. Dunlap, A. M., and Schuknecht, H. F.: Closure of Perforations of the Tympanic Membrane, *Laryngoscope* 57:479-490, 1947.
10. Kobrak, H. G.: *J. Acoust. Soc. of America* 13:179, 1941.
11. Kobrak, H. G.: *J. Acoust. Soc. of America* 15:54, 1943.
12. Kobrak, H. G.: *J. Acoust. Soc. of America* 19:328, 1947.
13. Kobrak, H. G.: *Transactions American Academy of O. & Co.*, p. 708, 1950.
14. Kovats, A.: *Wisconsin Medical Journal* 49:391, 1950.
15. Lebo, Charles P., and Palermo, Amiel L.: *ANNALS OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:1027, 1948.
16. Leschevin: Cit. by Haugh *Das Künstliche Trommelfell* Münschen Theodor Ackermann, 1889.
17. Nasiell, V.: Demonstration of Application of Artificial Membranes According to New Principles, *Acta Otolaryngology* 20:432-443, 1943.
18. Okuneff, W. N.: *Monatsschrift für Ohrenheilk.* No. 1, Berlin, 1895.
19. Pohlmann, A. G.: *Arch. Otolaryng.* 37:628, 1943.
20. Pohlmann, A. G.: *ANNALS OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 56:647, 1947.
21. Pohlman, Max: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:647, 1947.
22. Pohlman, Max: *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 57:483, 1948.
23. Steinmann, Erich P.: Über die Verwendung des Cellophans für Trommelfell prothesen, *Praxis* 34:46, 1945.
24. Toynbee, Joseph: On the Use of an Artificial Membrane Tympani in Cases of Deafness, London, 1857.
25. Tröltzsch, Anton F.: *Lehrbuch der Ohrenheilkunde* Leipzig, 1881.

"CLOCKING" OF THE NARIS AS AN AID IN
DEMONSTRATING RHINOPLASTY

A. P. SELTZER, M.D., ScD.

PHILADELPHIA, PA.

"Clocking" of the naris in plastic operations about the base of the nose contributes to a clearer understanding of the lines of incision and of the placement of sutures in the limited space occupied by the columella and the alae than can be conveyed by a simple statement. This visual method of demonstrating a somewhat difficult surgical technique has proved to be an aid in class and other similar demonstrations. It leaves no place for doubt about exactly what is being done and where.

The nose is generally described as a pyramid, but this image can be taken only with some reservations, and not with the precision of mathematics, since hardly any two noses are ever exactly alike.

The base of the nose is concerned here, where one sees a more or less flat surface in which there are two roughly oval openings, the nostrils. These openings, or nares, are of special significance from more than one viewpoint. Their function is of great importance to the body, since they are the entrances of the normal passageways for respiration, and the amount of air admitted is of significance to general health. If too much passes inward there is a disturbance of the dynamics of tidal air within the cavity of the nose, with consequent alteration of its physiology. Similarly, if the openings are restricted, the amount and force of the inhaled air is not sufficient to provide adequate aeration of the lungs, and general health again suffers.

Considered anatomically, the base of the nose consists of several parts. Below, it is in relation to the upper lip, with which it forms the nasolabial angle, and the skin of the lip is continued through the nostril to form the floor of the nasal vestibule, just inside the nares. The sides, or lateral borders of the nares are formed by thin cushions of skin and its underlying connective tissue layer, the alae,

From the Department of Plastic and reconstructive Surgery of St. Luke's Medical Center, Philadelphia.

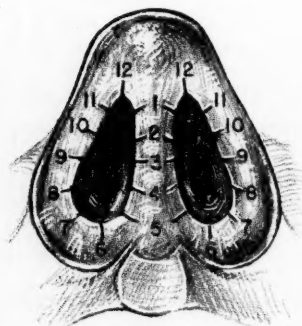


Fig. 1.—Illustrates the method of clocking the nares, clockwise and counter-clockwise, as an aid in demonstrating the exact position of surgical incisions about the nares.

which together form definite prominences on either side of the nose, where they are continuous with the skin of the outer surface of the nose, as well as that of the adjacent cheeks. The nostrils are separated by a relatively narrow strip of skin, the columella, which covers the lower border of the cartilaginous septum, and with the underlying layer of loose fibrous tissue is known as the movable septum (septum mobile). The upper portion of the base of the nose, as seen from below, is made up of the skin and subcutaneous tissues of the tip of the nose, which surround the anterior angles of the nares; a part of the nose of particular importance to the rhinoplastic surgeon.

One of the more difficult, and equally important conditions of the base of the nose is that which is present in complete hare-lip. This state of maldevelopment may involve the floor of the nose on one or both sides. There is usually a great degree of deformity of the nostrils in either case, caused by the cleft in the lower curve of the nostril, and by the abnormal shortness of the columella. To correct the anomaly surgically means closing the cleft or clefts, and rebuilding the nostrils so that they are alike, if the cleft is on one side only, or so that they are equal and normal in appearance, when both sides of the nasal floor are affected.

When the nostrils in an otherwise normal nose are grossly unlike in size and shape, they are made to correspond, by reducing the size of one side, or enlarging that on the opposite side. The choice of the method must depend upon which operation will create the most esthetic final effect.

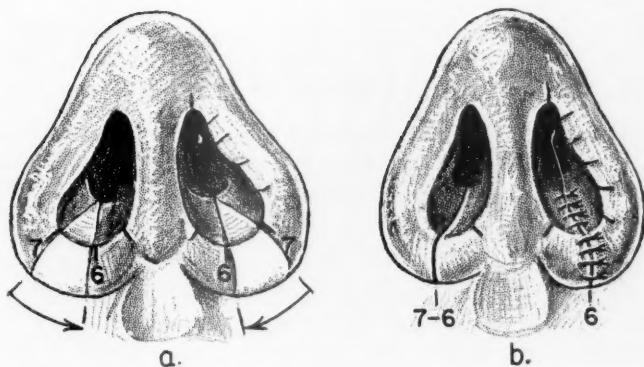


Fig. 2.—(a) For reducing the size of the nares, a wedge of tissue is removed from both sides of the base of the nose between six and seven o'clock. (b) Shows the approximated surfaces at six and seven o'clock with sutures in place, thus reducing the size of the nares.

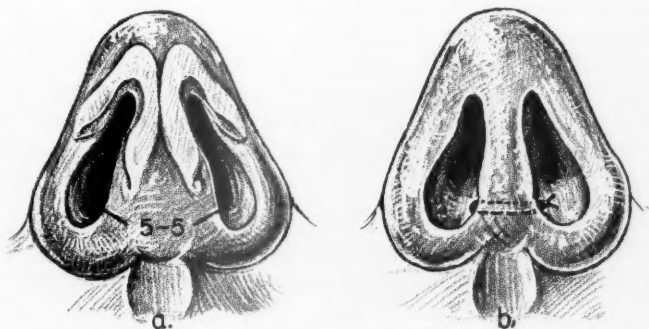


Fig. 3.—(a) Schematic view of the subcutaneous appearance of the basal surface of a nose with excessive width of the columella. Reduction of the extreme width of the columella is made by the surgical removal of the necessary amount of subcutaneous fatty tissue, with the surfaces approximated at five o'clock. (b) Shows the five o'clock suture in place in the surgically narrowed columella.

In the case of a columella that is too wide, there may be interference with the optimum amount of air entering the nasal cavity, or it may be only that this excess width should be corrected, in order to create a more esthetic effect. This operation for narrowing the columella can not only assure a more physiological airway, but at the same time improve the appearance of the nose.

Skin grafting may be required in some of these operations, and may be the case particularly when enlarging the side of the nostril, lengthening the columella, or reconstructing a deformed or scarred ala.

The nose is preeminently a frequent seat of injury in all types and degrees of accident, and extreme delicacy of operative procedure and accuracy of technique are of utmost importance from the standpoint both of the surgeon and of the patient; to this end, a definite means of indicating exactly where each step in the process of repair is placed is unquestionably desirable.

For class teaching or for other similar demonstrations of operating on the base of the nose, the writer has found that this clocking method is a definite aid in creating a clear visual image for the observers of the operations.

The scheme for definite localization used here is the same as that employed in many other relations, except for this purpose the two nostrils are clocked in opposite directions. On the left side, as seen by the operator, the progression is from left to right, or clockwise, 12 being as usual at the top; while on the right side, the figures are arranged counter clockwise. The clocked figures are used with equal ease whether the operation is concerned with only one side of the nose, or with both. Along the columella, the same figures correspond on both sides. Where both nostrils are included in the operation, it is evident that the designation of "left" or "right" must be included in connection with the figures.

It is hoped that others may find this simple aid to demonstration helpful in the course of demonstrating plastic operations on the nose.

2104 SPRUCE ST.

THE SURGICAL TREATMENT OF LARYNGOCELE

WITH REPORT OF A CASE

JOHN J. O'KEEFE, M.D.

PHILADELPHIA, PA.

In that the history, anatomy, and general clinical characteristics of laryngocele were reviewed and discussed in a recent report¹ by this writer, and because several other articles^{2, 3} are available in the recent literature, all dealing ably with this unusual entity, and of easy access if there is need for, or interest in, such a statistical review, those data will be omitted in this report except wherein found pertinent to the discussion at hand. This report is concerned principally with the technic of surgical excision of laryngocele, and will limit itself to factors relative to that end.

Basically, all laryngoceles arise from, and are dilatations of, the saccus, or appendix, of the ventricle of Morgagni. They are classified, according to Lindsay,⁴ as internal when the cystic dilatation is contained within the larynx: as superior external when further dilatation allows the cyst to perforate the thyrohyoid membrane and appear as a swelling in the neck: and as a combined internal-external laryngocele—a sort of dumb-bell configuration—one cyst contained within the larynx, and joined by means of an isthmus through the thyrohyoid membrane, with another cyst lying in the structures of the external neck. Because of these variations in position, several operative procedures have been advocated, each devised for a particular purpose.

It is axiomatic that laryngoceles sufficiently large to produce symptoms, or evidencing infection, should be excised. It is accepted too, that the technic of operation depends on the type or position of cyst present. Currently, the consensus^{3, 4, 5} is that for the external superior type of cyst, the approach is made through an external lateral neck incision, freeing the sac and stalk by blunt dissection, and amputating at the perforation in the thyrohyoid membrane. This is well illustrated in the reports by Shambaugh⁸ and Richards.³ For the internal type, it is argued that the best approach is by way of a thyrot-

From the Department of Laryngology and Bronchoesophagology, Jefferson Hospital, Philadelphia.

omy, either median, or some variation of lateral, again bluntly dissecting the sac free, and amputating as close to the ventricle as possible. Lewis⁹ first recommended the splitting of the thyroid cartilage in front of its superior horn as the best means of approach for excision of the stalk of the external laryngocele. This approach was modified by New^{6, 7} in his description of lateral thyrotomy for removal of the internal variety of laryngocele. For the combined type, as Lindsay⁴ stated: "... the surgical treatment is complicated inasmuch as removal of the external part of the sac must be combined with an intralaryngeal operation to remove the internal laryngocele."

The rationale for the mechanics of these various operative technics is not a subject of dispute: however, in their summation, an argument will be presented and a method described that appears to be anatomically and surgically adapted for the suitable excision of all types of laryngoceles.

REPORT OF A CASE

P. C., a white male, aged 47, was referred because of hoarseness and dyspnoea, and the presence of a globular mass in his larynx. The patient stated that his hoarseness had been present for more than four years, but that it had become progressively worse in the past year. His dyspnoea was first noticed about six months ago, but likewise had become markedly worse in the past few months.

Mirror laryngoscopy revealed the presence of a large globular swelling involving principally the left ventricular band, and overhanging the glottis so that the true cords could not be visualized. There was no palpable evidence of tumefaction in the neck, but there was slight to moderate interference with the mobility of the larynx. At direct laryngoscopy, no signs of ulceration or granulomatous masses were found.

Roentgen study of the neck illustrated a well-defined cystic tumor involving the left side of the larynx, which cyst was seen to balloon with air as the patient practiced the Valsalva procedure. The obvious diagnosis was laryngocele, internal type.

The patient was prepared for operation, and under endotracheal oxygen-ether anesthesia, a horizontal incision was made approximately a finger's breadth beneath and parallel to the ramus of the mandible, and extending from the midline of the neck to the anterior border of the sterno-cleido-mastoid muscle. The skin flaps included the platysma muscle. The anterior belly of the omo-hyoid was bisected,

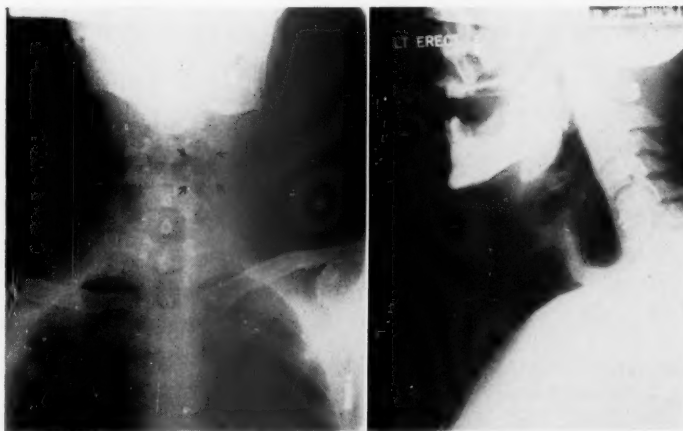


Fig. 1.—Postero-anterior and lateral films of the neck illustrating the presence of a large air cyst of the larynx, and the value of the valsalva procedure.

allowing full vision of the thyrohyoid membrane area. This membrane, with its overlying thyrohyoid muscle, was carefully incised in the same plane as the skin incision, and freed from the upper border of the thyroid wing. As its fibres separated, the dome of the laryngocele became apparent. Then, using cotton-tipped applicators—a technic described by New^{6, 7}—the cyst was gently and easily separated from its contiguous structures down into the substance of the ventricular band. Tilting of the thyroid cartilage, already made mobile by the sectioning of the thyrohyoid muscle and membrane, greatly facilitated in this dissection, and eventually allowed for amputation at the level of the ventricle. Amputation was made between double ligatures, and closure of the wound completed without drainage. No tracheostomy was necessary. The wound healed by first intention, and the patient was discharged on the sixth post-operative day. At the time of writing, three months later, the configuration of the larynx is well within normal limits, there is no evidence of recurrence of the cyst, and the patient is relatively asymptomatic.

The histologic report states that the cyst is lined by a respiratory type epithelium, its walls are infiltrated by chronic inflammatory cells, and there is no evidence of malignancy in any of the sections studied.

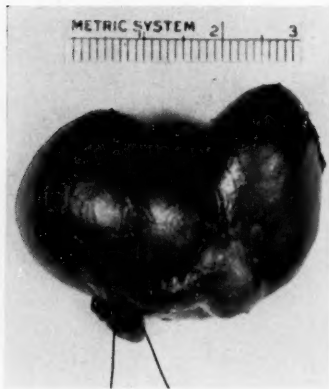


Fig. 2.—Photograph of cystic mass excised.

COMMENT

Opinion is unanimous^{3, 4, 5} regarding the surgical approach for excision of the external type of laryngocele. However, after reviewing the occasional cases reported,^{4, 6, 7} and drawing from a meager personal experience,¹ it is submitted as practical, to resect the internal variety—as well as the internal portion of the combined type—by way of a similar external operation.

Several factors play a significant part in the development of this method. The role of the endotracheal tube, which not only affords easy and adequate anesthesia, but insures the integrity of respiration during operation, is of paramount importance. Because of it one is allowed to manipulate the larynx, put traction on the cyst and its adjacent structures, and all the while be completely free of concern for the laryngeal airway. New^{6, 7} has contributed a further nicety of technic in his description of the use of cotton-tipped applicators as dissectors of the cyst. These are surprisingly effective and gentle, and do much to insure against rupture of the cyst; and removal of an intact or unruptured cyst is proof of complete removal. Too, it gives the operator a pleasing sense of superior skill, even though he knows it to be unwarranted on this score. Lastly, the procedure of mobilizing and tilting the thyroid wing downward appears to afford sufficient exposure of the cystic mass to effect complete dissection and amputation. If necessary, and rather than resorting to an intralaryngeal operation, one has recourse to split-

ting the thyroid ala, or resecting its posterior portion, as previously defined, in order to follow the cyst or its stalk down to the ventricle.

SUMMARY

The surgical technics for the various types of laryngoceles are considered, and through the reporting of another case of internal laryngocele, a method is reported that appears to be suitable for the resection of all varieties.

255 SOUTH 17TH STREET.

REFERENCES

1. O'Keefe, John J.: Laryngocele, *Arch. of Otolaryngol.* 54:29 (July) 1951.
2. Keim, W. Franklin, and Livingstone, Robt. C.: Internal Laryngocele, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 60:39 (Mar.) 1951.
3. Richards, Lyman: Laryngocele, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 60:510 (June) 1951.
4. Lindsay, J. R.: Laryngocele Ventricularis, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 49:661 (Sept.) 1940.
5. Allman, C. H., and Cordray, D. P.: Laryngocele, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 51:586 (Sept.) 1942.
6. New, G. B.: Treatment of Cysts of the Larynx, *Arch. Otolaryng.* 36:687 (Nov.) 1942.
7. New, G. B.: Congenital Cysts of the Tongue, the Floor of the Mouth, the Pharynx and the Larynx, *Arch. Otolaryng.* 45:145 (Feb.) 1947.
8. Shambaugh, cited by Lindsay.⁴
9. Lewis, cited by Allman and Cordray.⁵

LXXXVII

SURGERY IN INTRA-ORAL CANCER

MAURICE F. SNITMAN, M.D.

CHICAGO, ILL.

The attitude of the profession at large toward the treatment of intra-oral cancer is well exemplified in the statement by Blair "there is an uncertainty common in the profession today, an uncertainty that is at this time responsible for the more fatalistic antagonism that hampers therapy of the face and mouth cancer."

Prior to the advent of irradiation, at the turn of the century, cancer of the oral cavity was treated surgically and with comparatively poor results. The hope of increased curability following radiation therapy has not been realized. Consequently, surgery again is becoming the treatment of choice. Impetus to this modality has been provided by improvements in surgical technic and anesthesia, antibiotics, and a greater understanding of the importance of fluid and electrolytic balance. Additional measures such as parenteral and catheter feedings and tracheotomy have contributed greatly in this cause.

Cancer of the tongue, floor of the mouth and lower gingiva metastasize rather readily. In many instances accordingly, the treatment of the metastatic lesions depended on the sterilization of the primary. When irradiation of the primary was employed, active management of the metastases had to await the control of the primary and, in not a few cases, the involved nodes became inoperable during this waiting period. When surgery of cancer of the oral cavity and metastases was undertaken, it usually consisted of some form of removal of the primary and radical resection of the node-bearing area. This was too often destined to be inadequate because the intervening tissues of the mouth were left intact and recurrences there were the rule. In 1902, Polya and Nauratil pointed out that in about 50 percent of normal individuals, the lymphatics from the tongue and floor of the mouth pass through the periosteum of the mandible on their way to lymph nodes in the digastric triangle. This explained the frequency of attachment of metastases and primary growths to the jaw, emphasizing the need for resection of the body of the mandible and its periosteum whenever the primary growth originates or grows adjacent to it.

TABLE 1.—EXTERNAL CAROTID ARTERY LIGATION AND TUBE FEEDING IN ALL CASES.

AGE SEX	CASES				SURGERY	PROGRESS
	SITE	NODES	PREVIOUS THERAPY			
58 WM	Left lower gingiva. Tongue free.	-----	None.	9-20-51. Hemimandiblectomy with floor of mouth and suprahypoid dissection. Tracheotomy.	No evidence of disease. Speech and deglutition excellent.	
F.N.						
63 WM	Right side of tongue near base, adjoining anterior pillar and floor of mouth. No fixation of tongue.	Submaxillary?	None.	11-23-51. Resection right side of tongue, floor of mouth and suprahypoid dissection. Nodes negative on frozen section.	Healed. No evidence of disease.	
FLW						
67 WM	Right side of tongue posteriorly with involvement of floor of mouth, anterior pillar and floor of tonsil fossa. Tongue fixed.	-----	External irradiation. Constant pain.	12-13-51. Hemimandiblectomy, right side of tongue, floor of mouth, anterior pillar and floor of tonsil fossa. Radical neck. Tracheotomy.	Convalescing. No pain. External fistula. Healed.	
JNA						
56 WM	Right side of dorsum posteriorly. Some fixation of tongue.	-----	March 1951. Radon seed implantation, followed by forty pound weight loss, associated with severe constant pain.	12-28-51. Right side of tongue, floor of mouth, with suprahypoid dissection through mandible splitting.	Excellent. No pain.	
JAC						
55 WM	Left side of tongue, with involvement of anterior pillar and floor of mouth.	-----	None.	1-3-52. Hemimandiblectomy. Left half of tongue, floor of mouth. Suprahypoid dissection. Tracheotomy.	Excellent post-operative progress.	
FB						

In the literature of the last few years, a combined operation for unilateral malignancies of the tongue, floor of the mouth and mandible has been reported. Removal, *en bloc*, of primary and secondary tumor with the intervening lymph bearing tissue, is the sole feature of this comparatively new approach to the surgical management of intra-oral cancer. This removal, in continuity or monobloc procedure, finds its parallel in the Myles abdomino-perineal resection for carcinoma of the rectum, the Halsted radical breasts amputation, laryngectomy, parotidectomy and thyroidectomy combined with a radical neck dissection.

Five year cure rates are not as yet available. However, it is definitely felt that this method of composite surgery has given most encouraging results. The improved immediate result is demonstrated by absence of the almost constant excruciating pain that follows, in many instances, irradiation in one form or another. The personal observation of such pain-racked patients over a number of years will, in itself, lead to the ready acceptance of surgical extirpation where the lesion is totally resectable. It is gratifying that the operation, under our present pre and post operative management, is well tolerated. The absence of pain and the relatively minor hindrance to speech and deglutition are most surprising.

SURGICAL COMMENTS

The extent of the composite surgery will depend on the degree of involvement of the tissues of the floor of the mouth and the presence or absence of cervical adenopathy. Thus (a) when an adequate amount of normal floor is present between the lesion and the medial surface of the mandible, the latter is not removed. (b) In all cases without cervical adenopathy the bloc includes a suprahyoid dissection. (c) When the mandible can be spared, removal of the primary lesion, intervening lymph-bearing tissues and cervical dissection may be performed through a mandible splitting or pull-through technic. (d) Where the tissues of the floor of the mouth are fixed, the primary lesion, mandible, floor of mouth and cervical tissues are removed *en toto*. (e) Prophylactic radical neck dissections are not performed. (f) External carotid artery ligation in all cases. (g) Levine tube feeding postoperatively.

Abstracts of five patients' histories are tabulated. These illustrate the variations in surgical procedure. In all cases, the principle of the monobloc is adhered to.

CONCLUSION

We call your attention to an improvement in the management of intra-oral cancer by the surgical *en bloc* removal of the primary

lesion, together with the regional lymph nodes and intervening lymph-bearing tissue. It follows the fundamental principles laid down by Halsted fifty years ago, when he described the radical mastectomy. This operation has the advantage, in that the lymph bearing area can be removed simultaneously with the uncontrolled primary.

Improvement in surgical technic, pre- and postoperative care have made these radical measures possible, with a minimum of operative mortality and have given distinct promise of better results.

LXXXVIII

OBSTRUCTION OF THE AIR PASSAGES

WENDELL A. WELLER, COLONEL, MC, U. S. ARMY

SAN FRANCISCO, CALIF.

The early recognition of obstruction in the air passages is of utmost importance. Chevalier Jackson¹ stated: "There is only one route by which oxygen may be gotten into the tissues: namely the blood stream; and there is only one way, as yet known to medical science, by which oxygen can be gotten into the blood stream and that is through the walls of the air vessels of the lungs." Prompt and aggressive action to establish an unobstructed airway is imperative. Close cooperation between the laryngologist and other specialists is required. This problem is not new and confronts all physicians, civilian and military. Galloway² stated: "The importance and serious implication of anoxia is not always appreciated, especially under the stress of emergency." Some degree of anoxia is frequently unsuspected and untreated.

Obstruction may be caused by many conditions and associated with us many more. The upper or lower air passages may be involved separately or in combination. An accurate diagnosis should be made if possible.

Asphyxia.—Grodins, and his associates³ indicated that asphyxia is composed of three elements: anoxia, hypercapnia and acidemia. The intensity of the effects upon the body of reduced oxygen tension and increased carbon dioxide in the blood is influenced by the abruptness of onset of the anoxemia, its degree, duration and the general physical condition of the body.

Anoxia.—Anoxia, anoxemia, hypoxia, hypoemia, oxygen lack or oxygen want is the failure of the body tissues, for any reason, to receive an adequate supply of oxygen.⁴ Anoxia may result from four chief causes:

1. The anoxic type of anoxia results from defective oxygenation of the blood in the lungs.

Presented as part of a Symposium on Military Medicine at Murphy Army Hospital, Waltham, Massachusetts, June 23-25, 1952.

2. The anemic type is caused by hemorrhage or anemia from any cause, carbon monoxide poisoning, and poisoning by nitrates and chlorates. All lower the oxygen capacity of the blood.

3. The stagnant type is characterized by slowing of the movement of the blood through the capillaries. This occurs in heart failure, obstruction of the venous return from a part (local) anoxia and in shock.

4. The histotoxic type as in cyanide poisoning, where the respiratory mechanisms of the tissues are poisoned.

The anoxic type of anoxia may be produced by low oxygen tension in the inspired air, by abnormalities of the pulmonary mechanisms or by obstruction of the air passages. We shall consider the anoxic type as produced by obstruction of the air passages.

The effects of anoxia include an increase in capillary permeability, pulmonary edema, damage of the central nervous system and cardiac tissues.

Hypercapnia.—Hypercapnia is an excess of carbon dioxide in the blood. Obstruction of the air passages lowers the tidal air. This reduces the diffusion rate of oxygen into the blood, and also the diffusion and elimination of carbon dioxide from it. There is a resultant increase of carbon dioxide in the blood. Two principle effects result.

First, the narcotic effect of the gas itself, as Seevers⁵ points out, may be even more disastrous than those of anoxia. Symptoms attributed to hypercapnia include headache, restlessness, apprehension and disorientation in the early stages. High concentrations produce narcosis, anaesthesia, respiratory depression and circulatory collapse.

The second effect of hypercapnia is due to the reduction of the pH of the blood by the retention of free carbon dioxide. This produces respiratory acidemia. The carbon dioxide combining power of the blood is high.

The Upper Air Passages.—The upper air passages include the nose, nasopharynx, oral cavity and hypopharynx. These and adjacent structures may play an important part in respiratory obstruction. Severe infections, especially of the soft palate, pterygomaxillary, pterygopharyngeal or parapharyngeal region, Ludwig's angina, abscess of the tongue, or tonsillitis with extreme swelling, tumors, trauma, foreign bodies, blood and secretions are the chief etiological agents which cause encroachment upon this portion of the air passage. Edema of the tongue and palate or floor of the mouth usually

gives some warning of onset but may become emergent in a few minutes. Jackson and Jackson⁶ have stressed the lingual death zone in asphyxia.

Severe trismus, either from adjacent infection or from tetanus, a dangerous obstacle in establishing a satisfactory airway. Maxillo-facial fractures and gunshot wounds of the maxilla or mandible, which require wiring of the teeth for fixation of the bony fragments, are in the same category. Manifestations of high respiratory obstruction are dyspnea and retraction of the suprasternal, supraclavicular, intercostal and epigastric regions. These signs are pathognomonic.

The Lower Air Passages.—The lower air passages include the larynx, trachea, bronchi, and the smaller divisions of the pulmonary tree.

The larynx is the entrance to the lower air passages and is one of the sites for the initiation of the cough reflex. In this physiological role it is the "watch-dog of the lungs" (C. Jackson). Its cough reflex prevents foreign material from entering the trachea. Obstruction of the airway in the larynx, the subglottic area or the upper tracheal region produces a respiratory difficulty known as high obstructive dyspnea which is characterized by suprasternal, supraclavicular, intercostal and epigastric retraction.

The larynx is subject to infections, tumor and trauma. Any of these conditions which encroach upon the airway usually cause laryngeal stridor which demands immediate investigation.

Obstruction of the bronchi is caused by foreign bodies, benign and malignant new growths, enlarged peribronchial lymph nodes in tuberculosis or carcinoma, congenital formations, mucosal swelling in inflammations, granulation tissue, membrane as in diphtheria, blood clot and thick secretions. The effect on the pulmonary tissue beyond the point of obstruction varies upon the degree of blockage and may result in pulmonary atelectasis, obstructive emphysema or a "drowned lung."⁷

Obstruction of the lower air passages by tracheobronchial secretions has been called secretional obstruction. My preference is the term "tracheo-bronchial stasis." Increased tracheobronchial secretions and stasis are caused by any condition which will interfere with *coughing* or *swallowing*. These are principally diseases, tumor or trauma of the central nervous system which may result in coma, severe general debility, or paralysis of the larynx, diaphragm and

intercostal muscles. Other causes are fractures of the ribs or cervical spine, pain or the fear of pain following chest or abdominal operations.

Excess secretions cause obstruction, edema, and spasm of the bronchioles resulting in scattered areas of atelectasis. This provides a fertile culture media for any bacteria present, and favors the development of pneumonitis. Further, if sufficient fluid is permitted to accumulate the patient may drown in his own secretions.

In the presence of coma, severe debility, or pharyngeal paralysis, the nasopharyngeal and salivary secretions, vomitus, feedings or blood collect in the pharynx and are aspirated. The patient, on each inspiration, sweeps some of this material through the larynx and into the trachea. Should these materials not be removed, either by positioning, suctioning or both, the tracheobronchial system gradually fills and an increasing degree of anoxia develops.

Galloway,⁸ Priest, Boies and Goltz,⁹ Strobel and Canfield,¹⁰ Galloway and Seifert,¹¹ have reported on recent epidemics of poliomyelitis and have stressed the signs, symptoms and treatment of the bulbar type. In this disease pharyngeal and laryngeal dysfunction may appear quite early. Accurate appraisal of these symptoms will aid in preventing the development of anoxia and hypercapnia from tracheobronchial stasis.

CLINICAL MANIFESTATIONS OF ANOXIA

1. *Hyperpnea* is abnormal exaggeration of the respiratory movements. The respiratory rate has been observed as high as 56 per minute in adults and higher in children.

2. *Dyspnea* indicates difficult and labored breathing. This symptom is usually early, but may not be recognized in a weak patient or in one who has had sedatives. There may be an audible stridor.

3. *Cyanosis* depends upon the absolute amount of reduced hemoglobin in the capillary blood. Certain general conditions may greatly effect this: shock, hemorrhage, anemia, polycythemia, emphysema and heart failure. Cyanosis denotes impending asphyxia.

4. *Retraction* of the suprasternal, supraclavicular, intercostal and epigastric regions is usually noted in high obstruction and may be very evident in children.

5. *Restlessness* may be very noticeable and is an important sign. In the very ill, or debilitated individual there may be only a "wild" rolling of the eyes, as if searching for help.

6. *Cough* which is "wet," continuous, paroxysmal, or hacking in character indicates retained secretions.

7. *Coarse rales* over the trachea are the most constant chest findings in tracheobronchial stasis. Wheeze and rattles may be present.

8. *Mental Symptoms*: early there is exhilaration or excessive confidence and antagonism or combativeness. Later, this is followed by depression, confusion, disorientation, irrationality, unresponsiveness, lethargy and finally coma.

9. *Physical findings* are variable but usually the pulse increases in rate and is often weak. The blood pressure is frequently low. The degree of shock influences these findings.

MEDICATIONS

The employment of sedatives is contra-indicated when respiratory obstruction is suspected. *Restlessness*, otherwise unexplained and especially in the young child, may be a prominent symptom of impending anoxemia. A child who requires the voluntary respiratory muscles to maintain proper oxygen tension will not sleep until exhausted. Depression of the respiratory center by sedatives, especially narcotics, is to be avoided. The use of sedatives may be followed by complete surrender of an overburdened respiratory mechanism. Atropine and other related drugs which increase the viscosity of oral and tracheal secretions should be avoided. They make the removal of the secretions more difficult. It is better to remove secretions by suction than attempt to diminish their quantity by medication.

General anesthetics are to be avoided, as a rule, in obstruction of the upper air passages. Should general anesthetics be indicated in multiple injuries, attention is first directed to the establishment and maintenance of an unobstructed airway.

TREATMENT

There are several methods of relieving obstruction of the upper respiratory tract. The prompt opening of the mouth and clearing away foreign matter is first. Placing the body in the prone position with the face to the side aids in clearing the hypopharynx. The head should be six to eight inches lower than the rest of the body. Blocking the jaws open is helpful. A mouth gag, such as the Jennings "Loktite," is easily maintained in place and aids in the introduction of instruments in uncooperative patients. Insertion of an

airway guards against muscular relaxation of the tongue. Dentures should be removed.

In severe laryngeal spasms or edema, more positive methods are required. Insertion of an endotracheal tube or a bronchoscope of proper size will provide a temporary airway. The endotracheal tube should be placed into the larynx by direct laryngoscopy. The rubber or plastic endotracheal tubes may remain in place for several hours in an emergency. For longer periods a tracheotomy is indicated to prevent damage to the intra-laryngeal structures.

Endotracheal aspiration of secretions by catheter is a valuable method of treatment. The catheter may be inserted directly through a laryngoscope or indirectly through the nasal passage.^{12, 13} Any of these methods at times present some difficulty.

A method herewith suggested is an indirect one. It has proven very effective and relatively simple to use.

The procedure is most easily accomplished by hyperextending the head over a roll of two folded bath towels. The hyperextended position causes the forward projection of the upper cervical vertebra. This projection deflects the tip of the catheter forward into the larynx. In the adult, a 16 or 18 French rubber catheter is introduced into the larger nasal passage and the tip advanced to the level of the larynx. The patient is instructed to open the mouth widely. The tongue is grasped with gauze and pulled firmly forward. This fixes the larynx and prevents swallowing. With the tongue held firmly the patient is instructed to breathe deeply. On inspiration the catheter is "popped" into and through the glottic chink. This causes immediate and usually severe laryngeal spasm and cough. The catheter is then attached to the tubing of the suction machine. The patient is encouraged to breathe deeply and to cough. Movement of the catheter up and down in the trachea stimulates the cough reflex. Aspiration is continued intermittently until breath sounds are heard in all portions of the chest and the operator is assured the secretions have been adequately removed. The procedure may be repeated every four to six hours, as required. The catheter may be introduced with the patient in an oxygen tent or in a croupette. Oxygen may also be administered through the other nostril. Internes and residents on routine duty shifts are trained to carry out this procedure as indicated.

Conscious patients are most often in the semi-sitting position. This method can also be used with patients in the supine position. It is equally effective in unconscious patients.

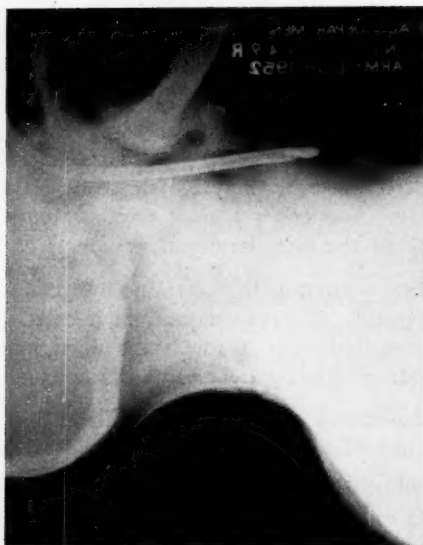


Fig. 1.—The tip of the intranasal catheter for tracheal aspiration entering the upper trachea. Head is hyperextended over 2 rolled bath towels and tongue held firmly fixing the larynx. Supine position.

Usually local anesthesia in the nose, throat or larynx is not required, in fact, its avoidance is preferable. The desired effect is a forceful and effective cough. The catheter is more efficient and less traumatizing to the mucous membrane of the trachea when a second $\frac{1}{4}$ " opening is made near the tip on the side opposite the usual opening. New rubber catheters are more easily inserted than older more flexible ones.

Clinical observations show a minimum of trauma to the larynx, even after repeated aspirations. A few petechial hemorrhages in the interior of the larynx have been noted. These may have been produced by the catheter or by the associated laryngospasm. See Figure 1.

Aspiration is not easy in infants and children. The larynx is exposed directly. The trachea and main bronchi are aspirated with a rubber tipped metal shafted suction tube such as devised by Samson-Davis.¹³

Inhalation anesthetics abolish the laryngeal and cough reflexes, inhibit ciliary action and stimulate excess secretions. The use of

routine adequate endotracheal suction during and after anesthesia clears the trachea and bronchi of accumulated secretions. This aids in reducing the post-operative pulmonary complications.

The use of a postnasal tampon for the control of hemorrhage from the nasopharynx may lead to difficulty should the patient close his mouth while reacting from a general anaesthetic. Care must be exercised to maintain an adequate oral airway.

Direct laryngoscopy and bronchoscopy provides visual examination of the air passages. Secretions are aspirated and other materials are removed as indicated. In post-operative massive pulmonary atelectasis when intranasal catheter suction has not been successful, early bronchoscopic aspiration is the treatment of choice. This assures maximum removal of secretions.

Tracheotomy is an operation which has been performed for centuries. It is still feared, due to the association of the serious conditions in which it is used. When properly performed, there are minimal complications. The tracheotomy tube should be placed in the midline through the third, fourth and fifth tracheal rings.

The indications for tracheotomy in obstruction of the upper air passages, larynx and upper trachea, are well recognized.

Conditions not so well recognized as producing tracheobronchial stasis and which may require tracheotomy are:

1. Infections as bulbar poliomyelitis, tetanus, botulism or diphtheria.
2. Drugs and poisons, anesthetics and barbiturates.
3. Central nervous system involvement: trauma, vascular accidents, abscesses or tumor.
4. Systemic nervous diseases involving the bulb including general paresis, tabes, gummas, disseminate sclerosis, amyotrophic lateral sclerosis, syringobulbia and glosso-labial paralysis.
5. Myasthenia.^{14, 15}

The performance of a tracheotomy may be simplified by the introduction of an endotracheal tube or a bronchoscope. This will transform the procedure into an orderly operation. An airway will be established, secretions can be removed and oxygen administered before the tracheotomy is begun.

Tracheotomy accomplishes three things. It by-passes the structures of the upper air passages and larynx, permits removal of ma-

terial aspirated from the nose and throat, and allows clearing of the lower pulmonary system of its own obstructing secretions.

Tracheotomies Performed.—During the past 17 months 30 tracheotomies have been performed at Letterman Army Hospital. The associated conditions are shown in the following table:

TRACHEOTOMIES L. A. H.
(1 January 1951 - 2 June 1952)

<i>Trauma</i>	
Gunshot wound, mandible	4
Gunshot wound, larynx	1
Mandibular fractures	3
Skull fractures (all in coma)	6
<i>Infection</i>	
Acute laryngotracheobronchitis	2
Bulbar poliomyelitis	3
Abscess, pterygopharyngeal and soft palate with severe trismus	1
<i>Tumors</i>	
Carcinoma of tongue (before interstitial radium)	1
Carcinoma of hypopharynx	1
<i>Pre-operative</i> Mandibular iliac bone grafts	4
<i>Post-operative</i>	
Neck surgery	2
Craniotomy	2
	30

DISCUSSION

The patients with gunshot wounds of the mandible and larynx received tracheotomies during their definitive treatment. The three mandibular fractures had other severe multiple injuries requiring general anaesthetics in their care.



Fig. 2.—Intranasal catheter for tracheal aspiration in place. Semi-sitting position. Note, hyperextension of cervical vertebra directs tip of catheter into the larynx.

The two cases of acute laryngotracheobronchitis were in small children, one 24 months and the other 25 months of age. Both children lived.

Of the three bulbar poliomyelitis patients two died, one within an hour and the other twenty-four hours following surgery. Minimal secretions were present in the trachea and bronchi at postmortem. The third patient lived despite a period of over two weeks of delirium and disorientation. Respirator treatment was required for two months.

The one patient with acute infection of the soft palate, pterygo-maxillary space and pharyngeal wall had severe trismus. He was critically ill, and cyanotic on admission. His infection had begun following the extraction of an upper left molar tooth (L-7). He had not responded to large doses of antibiotics. A tracheotomy was done. The palatal abscess was drained intraorally and the pterygo-pharyngeal abscess externally. A nasal feeding tube was placed. This patient made a rapid recovery.

The carcinoma of the tongue involved the middle and posterior thirds. The tracheotomy was performed prior to the insertion of interstitial radium needles. During the period the radium was in

CENTRAL NERVOUS SYSTEM TRAUMA
(Skull Fractures With Severe Brain Damage)

	DATE OF INJURY	ADMITTED	TRACHE- OTOMY	INDICATIONS	REMARKS
S.L.	21 Oct. 51	10 Dec. 51	2 Feb. 52	Coma, coarse rales, increased purulent secretions	Ablation frontal sinuses 14 Dec. 51. Internal hydrocephalus, coma. Died 15 Feb. 52. Autopsy, lung clear.
V.J.J.	17 Dec. 51	6 Jan. 52	7 Jan. 52	Coma, color ashen, coarse rales. Endotracheal tube 12 hours, large amount secretion.	Living, tracheotomy wound healed, mental deterioration.
A.T.C.	26 Jan. 52	27 Jan. 52	2 Feb. 52	Coma, increased tracheal secretions.	Living, severe mental deterioration, uses tracheotomy tube for occasional suctioning.
W.C.	2 Mar. 52	2 Mar. 52	8 Mar. 52	Coma, rales, right lower lobe consolidation.	Living, semi-comatose, bed patient, lungs clear.
B.A.	19 May 52	19 May 52	21 May 52	Coma, endotracheal suction not sufficient, cyanotic, respiratory rate 56. Large amount of secretions.	Living, semi-comatose, large amount tracheal secretions continue. Lungs clear by x-ray.
M.D.V.S.	30 May 52	30 May 52	2 June 52	Coma, Godel airway necessary. Hyper-pyrexia, increased tracheal secretions.	Living, semi-comatose, large amount tracheal secretions continue.

place, the patient became entirely dependent upon his tracheotomy due to edema of the tongue.

The carcinoma of the hypopharynx produced symptoms of respiratory obstruction and difficulty in swallowing.

Tracheotomies were done on four patients before the insertion of iliac bone grafts into the mandible. The immediate post-operative swelling of the floor of the mouth, the compression type dressing about the mandible, and the fixation of the jaws by intermaxillary wiring cause obstruction to the upper air passages.

Two patients following neck surgery required immediate tracheotomies. One, for hemorrhage into the mouth following the excision of a large thyroglossal duct cyst and the other for hemorrhage under a large neck skin flap.

In the two post-operative craniotomy cases obstruction developed from tracheobronchial secretions. One was in a small three month old hydrocephalic infant. Aspiration of thick mucus from the chest through an endotracheal tube gave only partial relief. Symptoms of high obstruction persisted. This child will not as yet tolerate removal of the tube. The other craniotomy was performed for an ependymoma of the fourth ventricle. Post-operatively this patient was unable to cough effectively due to a unilateral laryngeal paralysis.

The six patients with skull fractures with central nervous system damage, while different in the multiplicity of their other injuries, showed several common symptoms. All were in coma, all showed evidence of the effects of aspiration of nasal and oral secretions despite frequent suctioning, and all showed a change in the character of the tracheobronchial secretions, before tracheotomy was performed. The secretions increased in amount and became more purulent. One case developed consolidation by x-ray of the right lobe prior to operation. This cleared eventually. One case showed extreme hyperpnea and one hyperpyrexia.

It is evident from the post-operative course that these tracheotomies are life saving. They permit effective aspiration of the obstructing tracheal secretions. The nursing care is made simpler and is more efficient.

We have been particularly impressed by what has been accomplished by early tracheotomy in the care of comatose patients with tracheobronchial stasis.

Analysis of the 30 cases in which tracheotomies were performed shows only seven or 23% done for acute respiratory obstruction.

ANALYSIS OF 30 CASES

TRACHEOTOMIES DONE:

Group 1. As emergency procedures to relieve actual or impending respiratory obstruction

Acute laryngotracheobronchitis	2			
Abscess, pterygopharyngeal and soft palate with severe trismus	1			
Carcinoma of hypopharynx	1			
Post-operative neck surgery	2			
Post-operative craniotomy	1	Total	7	23%

Group 2. As prophylactic procedures to prevent possible respiratory emergency

Gunshot wound, mandible	4			
Gunshot wound, larynx	1			
Mandibular fractures	3			
Carcinoma of tongue (before interstitial radium)	1			
Pre-operative mandibular bone graft	4	Total	3	43%

Group 3. To relieve tracheobronchial stasis (Term is used to denote lack of expulsion of tracheobronchial secretions)

Skull fractures (all in coma)	6			
Bulbar poliomyelitis	3			
Post-operative craniotomy	1	Total	10	33%

The second group of 13 or 43% of cases were performed to prevent possible respiratory emergencies. This includes acute traumatic cases, many of which could not be handled safely without a positively established free airway.

In the third large group, 10 or 33% were performed to relieve *tracheobronchial stasis* before the patient's condition became critical from pulmonary complications. This group is evidence of the changing concept of the indications for tracheotomy in that there was a higher percentage of tracheotomies done than for those with acute respiratory obstruction.

SUMMARY

The causes, clinical manifestations and treatment of anoxic anoxemia in obstruction of the upper lower air passages are discussed. A

method for intranasal tracheal catheter aspiration is described. The reasons for the 30 tracheotomies performed at Letterman Army Hospital in the past 17 months are outlined. The analysis of the cases shows 23% of the tracheotomies performed for acute respiratory obstruction, 43% for prevention of respiratory emergencies and 33% for the treatment of tracheobronchial stasis. This indicates a changing trend as to the general indications for tracheotomy. The saving of lives by early tracheotomy in comatose patients with tracheobronchial stasis is emphasized.

LETTERMAN ARMY HOSPITAL.

REFERENCES

1. Jackson and Jackson: Diseases of the Nose, Throat and Ear, W. B. Saunders Company, Philadelphia, 1945, p. 475.
2. Galloway, Thomas C.: The Danger of Unrecognized Anoxia in Laryngology, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 55:508-513 (Sept.) 1946.
3. Grodins, Fred S., Lein, Allen, and Adler, Harry F.: Changes in Blood Acid-base Balance During Asphyxia and Resuscitation, *Am. J. Physiol.* 147:433-445 (Nov. 1) 1946.
4. Best and Taylor: The Physiological Basis of Medical Practice, 5th Edition, The Williams and Wilkins Co., Baltimore, 1951, p. 421-442.
5. Seevers, M. H.: The Narcotic Properties of Carbon Dioxide, *New York State J. Med.* 44:597-602 (Mar. 15) 1944.
6. Jackson, Chevalier, and Jackson, Chevalier L.: The Lingual Death Zone in Asphyxia, *New York State J. Med.* 34:1, 1934.
7. Morrison, W. W.: Diseases of The Ear, Nose and Throat, Appleton-Century-Crafts Inc., New York, p. 670-671.
8. Galloway, Thomas C.: Tracheotomy in Bulbar Poliomyelitis, *J. A. M. A.* 123:1096-1097 (Dec. 25) 1943.
9. Priest, R. E., Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 56:250-263 (June) 1947.
10. Strobel, R. J., and Canfield, N.: Tracheotomy in Poliomyelitis, *Arch. Otolaryngol.* 52:341-351 (Sept.) 1950.
11. Galloway, Thomas C., and Seifert, M. H.: Bulbar Poliomyelitis, *J. A. M. A.* 141:1-8 (Sept. 3) 1949.
12. DeWeese, David D.: Diagnosis, Prevention and Management of Post-operative Atelectasis, *Trans. of the Pacific Coast Oto-Ophthalmological Society*, 1951, p. 114.
13. Samson, Paul C., and Dugan, David J.: Tracheobronchial Toilet in Infant and Adult, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 59:738-748 (Sept.) 1950.
14. Von Leden, Hans: Newer Indications of Tracheotomy, *Trans. American Academy of Ophthalmology and Otolaryngology* 56:52-61 (Jan.-Feb.) 1952.
15. Galloway, Thomas C.: Discussion of No. 14, *Trans. Am. Acad. of Ophthalm. and Otol.* 56:63 (Jan.-Feb.) 1952.

LXXXIX

TREATMENT OF MALIGNANCY OF THE BUCCAL MUCOUS MEMBRANE, GINGIVA, SOFT AND HARD PALATE

JAMES W. HENDRICK, M.D.

SAN ANTONIO, TEXAS

This discussion of malignant tumors of the oral cavity includes buccal mucous membrane, gingiva, soft and hard palate. Carcinoma of the tongue, floor of the mouth, fauces, tonsil, anterior wall of the pharynx, and tumors of the jaws have been discussed in previous publications.^{34, 36}

INCIDENCE AND ETIOLOGY

Malignant tumors of the oral cavity comprise approximately eight per cent of all human cancer.²⁹ Any age may be affected with the greatest incidence occurring between the fifth and sixth decades. Men are more frequently affected than women in a ratio of eight to one. The most common etiological factor in the development of intraoral malignancy is chronic trauma, chemical or physical, or a combination of these. In a previous study of intraoral malignancy, it has been shown that benign lesions, i.e., chronic leukoplakia, broad base papilloma or leucic glossitis, preceded the malignancy in over 35 per cent of cases.^{17, 34} Tobacco, in any form, especially the incessant "cud" in the cheek,⁴ ill-fitting dentures which may produce and conceal trophic ulcers for long periods of time, or rough and ragged teeth, are causative factors of intraoral cancer. A rim of tartar around the gum margin of a tooth is considered a chemical agent which may excite malignancy. Sarcomas, adeno-carcinomas, and occasionally squamous cell carcinomas, may have origin in aberrant salivary tissue (Fig. 1A).^{9, 10, 34, 38}

Chronic oral infection, tobacco, and syphilis are considered causative factors in leukoplakia which is an epithelial hyperplasia. If these stimuli are permitted to persist, a definite malignancy may result; therefore, if the etiological factors, as well as premalignant lesions, are removed, malignancy may be prevented. According to Davis,⁶ oral cancer is frequent among Philippine women who chew buyo which contains black lime, betel nut and tobacco. Hart³⁴

reports 15 per cent of his oral cancer patients had positive serological tests for syphilis.

Squamous cell carcinomas involving the mucous membrane, gingiva, soft and hard palate are considered one group as they present similar characteristics and grow rather superficially from one area to another. These lesions remain superficial for sometime but if permitted to persist, eventually perforate the buccal fascia extending to the antrum⁷ and deep structures of the cheek. Those encountered on the posterior third of the gingiva or cheek near the angle of the mandible may extend on to the mandible, maxilla, or into the pterygoid fossa and are more serious than those elsewhere.

Epithelial malignancies arising on the mucous membrane of the upper or lower alveolar process, either in the neck of a tooth, around a tartar encrusted tooth, or on the buccal occlusional surface, may grow down into the cavity^{27, 36} where a tooth has been extracted to involve the jaw early. On the buccal mucous membrane of the cheek malignancies are more frequently encountered along the occlusional surface.

DIAGNOSIS AND CLINICAL BEHAVIOR

An adequate examination for intraoral malignancy requires careful and meticulous visualization of the entire intraoral cavity to note any ulceration or elevation with a smooth or hard base. Any thickened area should be carefully palpated with a gloved finger. In most instances the gross appearance of the cancer permits a diagnosis. The lesion may appear as an ulceration with an elevated, indurated, firm edge or as a papillary,³⁴ warty, fissured growth. Any lesion persisting in the mouth over three or four weeks should have adequate biopsy. Most intelligent patients will observe such lesions while they are small; on the other hand, it is not infrequent for clinic patients to appear with extensive ulcerations on the palate, gingiva, buccal mucosa, or large papillomatous growths almost filling the mouth. Carcinomas of the mucous membrane grow rapidly and the seriousness of the disease frequently is not appreciated by the patient.

Carcinomas occurring along the occlusional line of the buccal mucosa are fissured from trauma of mastication; infection occurs; the cheek becomes painful with induration of the tissues and loss of ability to masticate food resulting in malnutrition. As the tumor infiltrates the masseter, buccinator, and temporal muscles, their tendons become involved with trismus resulting. If the cancer is permitted to continue its growth, a fungating, ulcerating, sloughing

mass with a fetid odor perforates the cheek externally and extends into the surrounding intraoral structures to invade the jaw through tooth sockets.² The periosteum is a barrier to invasion for sometime but eventually the bone becomes involved; hemorrhage results from invasion of the internal maxillary or facial artery and/or vein and oral hygiene is practically absent in these cases.

When malignancy is detected on the soft or hard palate or gingiva, it is essential to carefully evaluate the extent of the growth as shown by roentgen examination to determine if there is invasion of the jaw or antrum.^{15, 36} The lymph nodes in the drainage area are carefully palpated to determine if they are involved; cancers of the gingiva, soft and hard palate and buccal mucous membrane do not metastasize with the same rapidity as those of the tongue, floor of the mouth, fauces and tonsil. Intraoral carcinoma remains localized to the mouth and lymph nodes in the drainage area, if it has advanced to them, for long periods of time without extending below the clavicle; consequently, it is important to diagnose these cancers early and eradicate the local lesion³⁴ and involvement in the lymph node drainage area, if present.

TREATMENT

The choice of treatment of intraoral malignancy depends on the location of the tumor, its histological classification, its size and extent, if the lymph nodes in the drainage area are involved, and age and general physical condition of the patient. When a diagnosis of intraoral carcinoma is made it is necessary to decide if it will be possible to cure the patient or if the treatment is only to serve as a palliative measure. A systematic outline of treatment should include hygienic care of the oral cavity before, during and after treatment, treatment of the primary lesion, and treatment of cervical metastasis, if present. Frequently the hygienic condition of the mouth in this group of patients is poor and if irradiation therapy is utilized, it should be directed toward preventing further discomfort to the patient. Any infected teeth or teeth in the field of irradiation should be removed by a dentist and the remaining teeth carefully cleaned before roentgen therapy is begun. Warm saline mouth washes used two or three times daily improve oral hygiene.

Surgery, electrosurgery, irradiation therapy, or a combination of these is used to eradicate intraoral cancer. Anaplastic cancers are more sensitive to irradiation therapy than the better differentiated carcinomas but since they metastasize earlier and more extensively, their prognosis is poorer. Squamous cell carcinomas of the lower



Fig. 1A.—Malignant mixed salivary tissue tumor of hard palate widely resected with electrosurgery. Metastasis to left cervical area five years later, which necessitated radical neck dissection. No recurrence after three years.

B.—Adenocarcinoma, upper right alveolus and maxilla.

histologic grades can be completely and permanently eradicated when it is possible to concentrate the radiant energy on the tumor; however, it must be appreciated that radiocurability and radiosensitivity are not synonymous. Cancers located on areas with good vascular beds³⁴ can be completely eradicated without danger to the tissue beneath the tumor but if the location is superficial over cartilage or bone, sufficient x-ray therapy essential to eradicate the tumor may result in a persistent painful ulcer or radio-osteonecrosis. Adenocarcinomas and mixed tumors of salivary tissue origin are radio-resistant and should be surgically excised;^{9, 10, 21, 37} melanomas are not sensitive to x-ray therapy.

Treatment of carcinoma of the upper gingiva and hard and soft palate has varied throughout the years to include electrosurgery, surgery, irradiation, or a combination of these. The recommended treatment for destruction of a primary superficial growth if it is under 2 cm in diameter is roentgen therapy and resection of lymph nodes in the drainage area if there is clinical evidence they are involved. Such lesions (Fig. 1B) are given 5000 to 6000 roentgens tumor dose through an intraoral cone. Treatment factors are: 140 KVP, 8 ma, $\frac{1}{4}$ Cu plus 1 Al filter, 24 cm TSD with a cone of sufficient size to adequately cover the lesion. Various types of cones are now available for intraoral roentgen therapy; the lesion can be well localized with an illuminating del Regato localizer. Lesions of

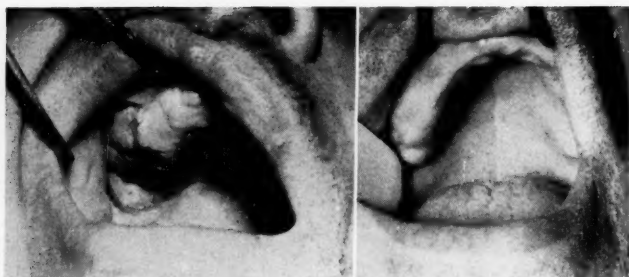


Fig. 2.—Extensive squamous cell carcinoma right maxilla extending on to hard palate with positive lymph nodes in drainage area.

B.—Appearance of tumor after intraoral x-ray therapy 6,000 r followed six weeks later by extraoral resection of superior maxilla and radical neck dissection. Clinically, this lesion has the appearance of complete destruction by radiation therapy but persistent cancer was found when right maxilla was resected. Prosthesis applied following operation. Good functional and cosmetic results. No recurrence of tumor.

this type can also be removed by electrosurgery; an incision is made with the electrotome around and well beneath the tumor giving a safe margin of 1 cm or more. There is good healing with a soft pliable scar following either of these methods of treatment of smaller lesions.

Cancers 1 cm to 2 cm in diameter which occur on the hard or soft palate or upper gingiva back of the central incisor teeth are difficult to treat with x-ray therapy through intraoral cones but are removed with electrosurgery or radium given in a leaded resinous applicator devised by Pyott and Ward²⁵ which may be repeated if necessary.

The soft palate is not infrequently the seat of mixed tumors of salivary tissue origin which undergo malignant transformation in 40 per cent of cases;³⁴ their removal is best accomplished by wide electrosurgical excision. If a large defect results, a prosthetic appliance is utilized which allows the patient to take adequate food, speak normally, and prevents food and liquids from entering the nasopharynx and nose.

Small cancers of the lower gingiva under 2 cm in diameter with no evidence of involvement of the mandible are treated with intraoral x-ray therapy as described above, electrocoagulation, or a Pyott applicator carrying radium. Irradiation therapy frequently results

in tissue changes which require electrosurgical excision of the irradiated area six to eight weeks later at which any persistent tumor is also removed giving a wide margin around and beneath it. It is seldom that cancer of the lower gingiva and mandible is detected early. The tumor is usually found to be extended on to the buccal surface and floor of the mouth or gingivobuccal sulcus to involve the mandible. These more extensive cancers require radical operative procedures through an extraoral approach after they have been given intraoral irradiation. This will be discussed in detail in malignancies of the tongue and floor of the mouth at a later date.

Larger lesions, $2\frac{1}{2}$ cm in diameter or over (Fig. 2), are first treated with irradiation therapy in the amount of 5000 to 6000 roentgens x-ray through an intraoral cone. In the event the lesion is too large to safely treat through a single port, the intraoral dosage is reduced to 3000 to 4000 roentgens and appropriate external ports are used; the number, size, and location of these vary with the location of the tumors. Usually two or three external ports are used. Treatment factors are: 260 KVP, 18 ma, Thoraeus filter equivalent to 1 mm Cu, 50 cm TSD with appropriate size ports. Daily intraoral treatments may be administered in doses of 300 to 500 roentgens and the external ports may receive 200 to 300 roentgens. Two alternate ports may be treated daily. Five to six weeks following administration of roentgen therapy, the treated area is widely excised with electrosurgery. It may be necessary to resect all or part of the superior maxilla and/or the hard palate, and into the antrum if it is involved. Immediately post-operatively the wound is packed with iodoform gauze. The wound heals by granulation and cicatrization during which time a suitable mouth wash is employed.

When it has been necessary to open the antrum, the patient may need to wear a temporary dental prosthesis to permit his immediate rehabilitation; this is replaced by a permanent one after the wound is entirely healed. In more advanced tumors of the upper gingiva, maxilla, hard and soft palate^{33, 34} which show invasion of the bone and extension into the antrum, nasoantrum, and lateral antral walls by roentgenologic examination, an extraoral surgical approach is utilized.

Extraoral resection of the superior maxilla, with or without the hard palate, (including removal of tumors involving the antrum) was formerly considered a formidable operation. With improvements in operative technique, anesthesia, use of antibiotics, and adequate blood from a blood bank, this procedure is now effected with low mortality and morbidity and high cure rates with good cosmetic



Fig. 3A.—Illustration showing line of skin incision of operative procedure for radical removal of carcinoma of upper gingiva, maxilla and maxillary sinus. Cheek turned back; dotted line shows area to be severed through maxilla and limbs of zygoma. The buccal fat is resected along with maxilla as it frequently harbors malignant cells.

B.—Superior maxilla and growth have been widely removed with infraorbital plate. Strips of fascia lata sutured medially and laterally will support the globe and prevent diplopia.

C.—Maxillary cavity and cheek lined with split thickness skin graft.

results. It is especially applicable in the treatment of adenocarcinoma arising in mucous and mixed salivary tissue with or without extension into the antrum, radio-osteonecrosis of the maxilla and hard palate, osteomyelitis, and squamous cell carcinoma involving the maxilla and antrum. The operative procedure used at this time which is performed five or six weeks following completion of administration of roentgen therapy is an evolution of techniques of Koenig,³⁴ Ward and Edgerton,³³ MacFee,¹⁹ and the author. Many complications which were formerly present in this procedure are no longer encountered, i.e., necessity for repeated dressings, postoperative hemorrhage, cosmetic defect of the operated side, diplopia resulting from sinking of the globe, and edema of the lower eyelid. Immediate skin graft is utilized to replace the lining of the antrum and cheek, a hallow-ball prosthesis permits the patient to converse normally with no difficulty in breathing or swallowing and allows adequate mastication of food. The hallow-ball type prosthesis carries teeth in the resected area of the jaw and is attached to the remaining teeth in the non-operated area.

Operative Technique. Intratracheal anesthesia using pentothal sodium intravenously, nitrous-oxide, oxygen and curare is utilized. The patient is placed in a mild Trendelenburg position with a pillow beneath the shoulders to prevent mucous and blood from entering the stomach and lungs. The pharynx is packed off with a moist gauze; the external carotid artery just above the lingual branch is divided to prevent troublesome hemorrhage during and after the operation.

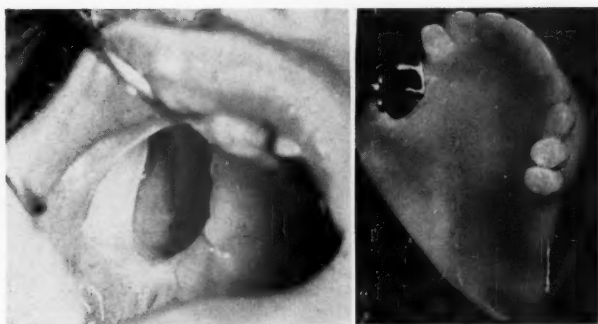


Fig. 4A.—Intraoral appearance following resection of superior maxilla. The split thickness graft is observed in this view.

B.—Prosthesis carrying teeth permits patient to swallow and converse normally.

The operative procedure is carried out through a Fergusson incision which begins at the vermilion border of the upper lip and extends up to the columella of the nose and around the ala on the involved side extending up to 2 cm below the inner canthus of the eye (Fig. 3A). The mucous membrane is divided along the gingivolabial sulcus as far around as the last molar tooth. If there is evidence the tumor has extended to the pterygoid fossa and involved the temporal muscle, the skin incision may be extended around laterally just below the orbit in a curved line with the concavity upward following the concavity of the orbital ridge to the malar bone then across toward the ear above the zygoma. It has been my policy to leave the fat pad of the cheek against the tumor to give a safe margin over it.

The superior maxilla is divided through the alveolar ridge by using a thin osteotome. A Gigli saw is used to divide the arms of the zygoma (Fig. 3B); the hard palate is split as far back as necessary and, if possible, a thin rim of bone is allowed to remain in the infraorbital ridge to support the globe. Occasionally it is necessary to remove this area of bone and in some instances the globe also. When the tumor has extended any distance posteriorly, it may be necessary to remove the most anterior part of the masseter muscle. If necessary, the pterygoid plate and muscles are included in a block dissection with the tissues in this area. The maxilla is gently loosened and removed with a thin osteotome and lion-jaw bone forceps. Histologic examination of any suspicious tissue is made during the

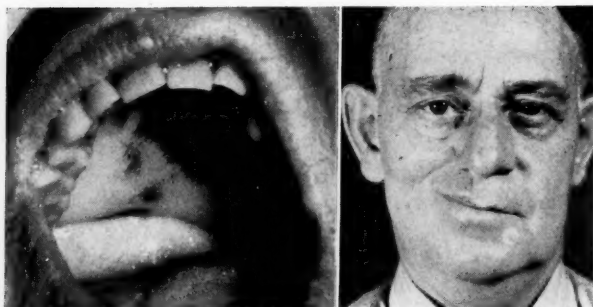


Fig. 5A.—Intraoral appearance with prosthesis in position.

B.—Cosmetic results following extensive resection of superior maxilla for extensive carcinoma.

course of the operation enabling the surgeon to remove all area involved. Electrocoagulation may be utilized for bleeding or destroying a suspicious area of tumor. The internal maxillary artery and vein will be encountered when the dissection reaches the region of the pterygoid fossa and will require ligation. Care is exercised to avoid injury to the cribiform plate which would produce a leak of cerebrospinal fluid and probable postoperative meningitis. When the infraorbital plate has been removed, a strip of fascia lata is placed just beneath the globe and sutured laterally and medially to give adequate support to the globe and prevent diplopia (Fig. 3 B).

During the operative procedure any tumor area which cannot be adequately removed by surgical measures, or any doubtful area of tissue may be treated with radium tubes in rubber cots left against the suspicious area sufficient time to give adequate therapy. The proper dosage of radium is calculated to give 6000 to 8000 gamma roentgens at 1 cm depth. The radium tubes are attached to strings of black silk brought out through the operative wound and strapped to the skin. The radium element is held in place by the iodoform gauze pack.

A split-thickness graft is removed from the anterior surface of the thigh with a Brown Electrodermatome and placed over the under surface of the cheek flap, care being exercised to cover the entire raw surface of the cheek, antrum, and pterygoid fossa. The graft is extended within 1 cm of the edge of the skin and sutured with fine catgut sutures to the mucosa of the cheek (Fig. 4A); several sutures



Fig. 6.—Adenocystic basal cell carcinoma involving right upper gingiva and maxilla. This type of tumor penetrates and infiltrates deeply. Apparently these tumors take origin in the mucous glands or the basal layer of the epithelium. They are comparable to adenocystic basal cell epitheliomas encountered in the skin but are more malignant. They are not very sensitive to irradiation therapy and require extensive surgical excision without preoperative preliminary irradiation therapy.

are placed over the surface of the graft to prevent collection of serum beneath it.

The cheek is replaced in its normal position and sutured with interrupted sutures of fine silk; the cavity is firmly packed with iodoform gauze which has been previously moistened with Peruvian balsam and a large pressure dressing is applied over the involved side of the face and cheek. The patient is carefully observed for several hours to prevent the aspiration of blood and mucous into the bronchial tree. The iodoform gauze is removed gradually during the first week following the surgical procedure and replaced by a temporary prosthesis as mentioned previously (Figs. 4B, 5A, and B).

Carcinoma of the Buccal Mucosa. Carcinoma of the buccal surface of the cheek requires more accurate and intensive therapy because this group of tumors grow more rapidly, invade the deep structures of the cheek more quickly,^{7, 13, 18, 21} and metastasize more widely than carcinomas of the gingiva, or soft and hard palate. Small and moderate size squamous cell carcinomas of the buccal mucous membrane under 2½ cm in diameter which have not invaded the underlying muscles may be adequately removed with intraoral roentgen therapy as described above allowing a margin of 1 cm or more around the tumor, or with the use of electrocoagulation allowing a wide margin around and beneath the tumor (Fig. 6). Either

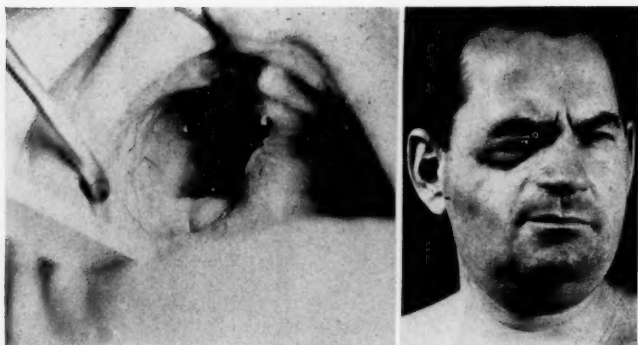


Fig. 7A & B.—Intraoral appearance following resection of superior maxilla case shown in Fig. 6. The tumor had penetrated the maxilla to the extent that it was essential to resect the infraorbital plate. Appearance of the split thickness graft lining the resected area.

B.—Cosmetic results following extensive resection of superior maxilla. There is slight drooping of the right globe which can be corrected by fascial support of the infraorbital area.

of these procedures leaves a soft and pliable mucous membrane without fibrosis of the underlying muscles.

Polya²⁴ described a variety of unique operations for removal of small and large mucosa malignancies with immediate closure by plastic flaps; however, irradiation given in adequate amounts permits such excellent results in the treatment of these smaller cancers that formidable operative procedures have been practically discontinued except when there is extensive involvement including perforation of the cheek and extension of the tumor on to the mandible or into the pterygoid fossa or maxilla.

Squamous cell carcinomas over 3 cm in diameter which have extended deeply to involve the buccal fat or invaded the masseter, buccinator, temporal, or internal pterygoid muscles and fossa are more difficult to eradicate. If trismus is not present and the mouth can be sufficiently opened, these cancers are best treated with intraoral roentgen therapy, 5000 to 6000 roentgens tumor dose, giving a wide margin around the tumor. When the muscles of mastication are involved and trismus does not allow the mouth to be opened sufficiently wide to permit introduction of an intraoral x-ray cone, extraoral roentgen therapy must be utilized. If there is evidence the tumor is still viable following this procedure, it is removed by electro-surgical excision giving a wide margin around and beneath the tumor.



Fig. 8A.—Large squamous cell carcinoma right buccal mucous membrane following inadequate treatment of leucoplakia of the cheek four years previously.

B.—Smaller squamous cell carcinoma involving buccal mucous membrane. This is a frequent position for buccal carcinomas.

If there is clinical evidence the lymph nodes in the drainage area are involved, a radical neck dissection is performed on the involved side. Since considerable infection is always present in cases of large fungating cancers, the lymph nodes in the drainage area, especially at the angle of the mandible and in the submaxillary group, frequently are enlarged which may be due to infection, malignancy, or both. If the nodes are enlarged from infection, they are usually tender and adherent and if involved from malignancy alone, are hard and in most instances, non-tender, and mobile. An endeavor is made to control the primary malignancy and discover the exact cause of lymph node enlargement before neck dissection is performed.

Jackson and New¹¹ report commendable results following the above plan of treatment in a group of buccal carcinomas. When a block neck dissection with positive lymph nodes is carried out, these authors stress the importance of making the treatment radical enough to completely eradicate the tumor; on the other hand, Martin and Pflueger¹⁸ prefer to treat such cancers with roentgen therapy and not perform a neck dissection unless metastases are found when the tumor is first encountered or if metastases develop at a later date. These authors defer neck dissection during the active treatment of the primary lesion, if irradiation therapy is employed.

Ankylosis of the mandible is a frequent complication following treatment of buccal carcinoma with radium needles. In these cases



Fig. 9.—Buccal carcinoma that has penetrated and perforated left cheek. Marked trismus is present. There is involvement of cervical lymph nodes. Treatment: Intensive x-ray therapy to the tumor, 6,000 roentgens, followed six weeks later with wide surgical excision with electrosurgery and radical neck dissection. Defect in cheek and lip covered with pedicle graft from acromioclavicular area several months later with good functional results. No recurrence.

which have been treated elsewhere, the author employs a submucous tenotomy severing the fibrosed muscle in three or four areas with a small tenotomy knife. If this procedure does not give satisfactory results, an incision is made through the ascending ramus with an osteotome, creating a false joint as suggested by Ward.^{34, 36}

Implantation of radium needles to treat cancer of the buccal mucosa is now obsolete. This method of therapy introduces infection into the deeper structures of the cheek through the path of the needles,^{14, 34, 35} results in extensive scarring and ankylosis of the jaw, and allows persistence of the tumor and recurrence in a high percentage of cases.

Large lesions which have infiltrated the buccal fat, perforated the cheek to involve the skin, or extended on to the lip or into the muscles of mastication, must be carefully evaluated to determine if the maxilla, mandible, or lymph nodes in the drainage are involved. Teeth in the immediate vicinity are removed and oral hygiene is meticulously carried out. X-ray examination of the mandible of the involved side, as well as the chest, is made before therapy is begun. As soon as the jaw is healed where the teeth have been extracted, the tumor is actively treated with roentgen therapy, intra- or extraoral, or both, giving a maximum tumor dose of 6000 to 8000 roentgens. Antibiotics are administered during this period to control infection.

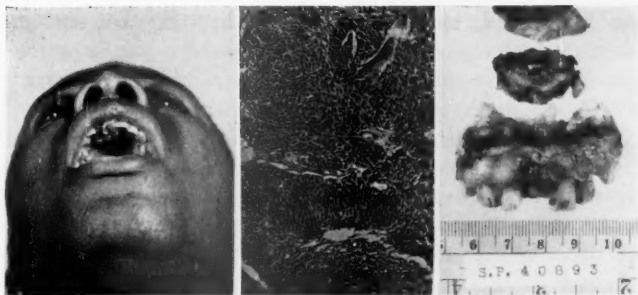


Fig. 10.—Malignant melanoma of alveolar ridge of eight months duration. Extensive resection of alveolar ridge. Patient died from metastasis several months later.

If the patient is unable to take adequate nutrition, a plastic nasal tube may be employed through which 3000 to 4000 calories of food with adequate vitamins are administered daily. Martin¹⁶ has developed a method whereby the patient feeds himself through a nasal catheter using a large syringe as frequently as necessary to permit adequate caloric intake.

Four to six weeks following completion of roentgen therapy, wide electrosurgical excision of the involved area is carried out, including all parts of the mandible, maxilla, cheek, lip, and skin. Part or all the mandible on the involved side may be destroyed by roentgen therapy. If there is clinical evidence of lymph node involvement in the drainage area and the condition of the patient permits, a neck dissection is performed at the same time the tumor is removed electrosurgically. If the neck dissection is necessary but must be deferred to a later date, the external carotid artery is ligated and severed above the lingual and superior thyroid branches. This is a precautionary measure to facilitate electrosurgical excision and prevent subsequent hemorrhage which is a frequent complication in extensive malignancies.

Radio-osteonecrosis or osteomyelitis may result from intensive roentgen therapy to extensive buccal carcinomas and it is essential in these cases to resect the involved area of mandible. If possible, a sufficient amount of mandible is left for stabilization; however, with the use of modern prostheses, one need not hesitate to resect the entire thickness and, if necessary, disarticulate the posterior fragment which only acts as a mechanical nuisance if allowed to remain.

The more extensive carcinomas which extend on to the mandible and destroy the soft tissues of the cheek, lip, and skin, and extend into the submaxillary area of the neck to involve the bone, on first examination, may appear hopeless but careful evaluation of the case,^{8, 19, 20} considering previous inadequate therapy which may have been administered, appropriate roentgen therapy, good oral hygiene, antibiotics, and adequate nutrition, may enable the patient to return in four to six weeks with the lesion markedly improved with only small areas of malignancy and granulating surface remaining. At this time the case is re-evaluated to determine the course of further therapy.

If it is determined the tumor can now be resected in its entirety, the following plan is carried out: all involved or suspicious tissue is removed with electrosurgery allowing a wide margin around and beneath the tumor. When possible, it is advisable to save the lingual or inner side of the mandible to serve as a scaffold for reconstruction of the jaw even though the outer table is frequently involved along with the soft tissues. In these cases the growth, periosteum, and hard cortical bone are removed with a rongeur and electrosurgery down to the cancellous bone, which permits better healing. When the entire thickness of the mandible is involved, it is removed, including a disarticulation if necessary. Present day prostheses permit wide removal of either the maxilla or mandible without resultant disfigurement. Kelly and Ward¹² described a method of sterilizing an involved area of mandible after resection of the soft tissues. Blair and co-workers⁵ described a similar technique. The dissection may extend down to include the submaxillary and suprahyoid area of soft tissue which may be involved by continuity of the growth or metastatic involvement of lymph nodes. If frozen section examination of involved lymph nodes reveals malignancy, a radical block neck dissection is performed if the condition of the patient permits. The external carotid artery is ligated to prevent hemorrhage at the beginning of the operative procedure. The defect in the cheek and mouth is closed, if possible, by bringing the tissues of the cheek and neck together which allows the patient to resume normal activities.

A long tubed pedicle graft is raised from the acromioclavicular area at the time of initial operation if the condition of the patient permits, or later if not, to cover any cheek, neck, or mouth defect. A pancake of sufficient size to cover the defect is made on the lower end of the graft and lined with a split-thickness graft taken from the thigh which will serve as an inner lining for the cheek and

mouth.^{7, 34, 35} By preserving the gutter between the mandible and tongue, the patient has better mobility of the tongue and is better able to masticate food properly, to drink, swallow, and carry on normal conversation.²⁷ The pedicle graft is not transferred to cover the defect in the cheek or mouth for six months to one year following surgery which permits any recurrence or persistence of the growth to manifest itself. In the past, before this method of management was utilized, occasionally recurrence or persistence developed under or around a graft and all the work would have been in vain as the growth would become extensive before it was recognized.

METASTASIS FROM CANCER OF THE BUCCAL MUCOSA,
GINGIVA, AND PALATE

A clear understanding of the lymphatic drainage of the mucous membrane of the mouth permits a more accurate examination for metastasis.^{34, 35} This was well reviewed in a previous communication and only a résumé is given here. The lymphatics from the posterior portion of the cheek drain to the parotid node; those from the anterior portion drain into the submaxillary nodes and the nodes at the angle of the mandible. The deep lymphatics of the cheek drain into the facial nodes; the lymphatics from the lower gums unite into several chains, pass over the outer surface of the mandible opposite the last molar tooth and enter the nodes in the submaxillary area. The lymphatics of the hard palate are continuous laterally with those of the upper gums and enter into several chains, pass backward in the midline of the palate to the level of the third molar tooth and separate in front of the anterior pillars of the fauces. From there they extend to the superior constrictors of the pharynx and enter the deep cervical nodes along the internal jugular vein above the posterior belly of the digastric muscle. Some lymphatics of the soft palate and nasal mucous membrane extend backward through the superior constrictors of the pharynx into the retropharyngeal nodes while others pass out beneath the mucous membrane of the posterior pillars of the fauces to the deep superior cervical nodes.

Metastases to the lymph nodes in the drainage area from primary malignancy of the soft and hard palate, upper gingiva, and buccal mucous membrane occur late in most instances. Metastases from the lower gingiva, tongue, floor of the mouth, and tonsils occur more extensively with greater rapidity.^{32, 34} Early metastasis from the lower gingiva is probably by way of the lymphatic channels which pass over the lateral side of the mandible permitting the floor of the mouth and lymph nodes in the drainage area to be invaded.

Richards²⁶ reports 50 to 60 per cent of cancers of the buccal surface of the mouth never metastasize at any stage. Jackson and New¹¹ report that 26 per cent of their series had metastases on admission, all of which were limited to the side of the neck on which the primary lesion occurred except one which was bilateral. Simmons,²⁷ reporting 387 cases of buccal mucous membrane cancer, states 68 per cent of his group had metastases to the lymph drainage area on admission. Martin and Pflueger¹⁸ report 56 per cent of their group had no palpable lymph nodes at the time of admission; however, 51 per cent of the entire group had metastases at one time or another; two patients in that series had bilateral metastases to the submaxillary area and two others had extension of the disease below the clavicle. My observations have shown that Grade I squamous cell carcinoma of the palate, mucous membrane of the cheek, and upper gingiva metastasize rather late;³⁴ however, the higher histologic grades of tumors, especially of the buccal mucous membrane of the cheek and lower gingiva, metastasize early in at least 40 per cent of cases.

Treatment of Metastatic Lymph Nodes. No active treatment of the lymph drainage area of the mouth is instituted in cases of cancer of the upper gingiva, palate, or buccal mucous membrane of the cheek unless there is clinical evidence of involvement of the lymph nodes, in which case a radical neck dissection is carried out on the involved side.³⁴ When the primary lesion is on the buccal surface of the cheek and has been eradicated by previous irradiation therapy, the lymph node dissection, when necessary, is carried well up over the lateral surface of the mandible. The more extensive cancers of the lower gingiva require radical dissection of the neck, including the jaw and soft tissues, which will be discussed in the Composite Operation for cancer of the tongue and floor of the mouth. Prophylactic x-ray therapy, which was formerly used by some in amounts of 1500 to 2000 roentgens given over large portions of the neck, has been discontinued and is totally obsolete as it has no value or merit whatsoever.

Irradiation therapy is advised for the treatment of metastatic lymph nodes in the neck in the following cases: (1) patients who have large single nodes who either refuse operation or are poor operative risks are treated with roentgen therapy using a cone giving 2500 to 3000 roentgens followed by implantation of radon seeds. The total dose should be 8000 to 10,000 gamma roentgens. (2) Patients with large inoperable masses of fixed nodes which may be reduced in size and become mobile as a result of roentgen therapy so

radical surgical excision can be carried out, are treated with 2500 to 3000 roentgens to the mass in broken doses through appropriate ports.³⁵ This treatment is followed by implantation of radium element needles or radon seeds administered in intratumoral doses of approximately 8000 to 10,000 gamma roentgens.

PROGNOSIS

In general, the prognosis of malignant tumors involving the gingiva, buccal mucosa, hard and soft palate, will depend on early diagnosis, histologic grade and extent of the tumor, presence or absence of metastasis at time of admission or subsequently, and adequate therapy, either roentgen therapy, electrosurgery, surgery, or a combination of these.

SUMMARY

Carcinoma involving the gingiva, palate, and buccal mucosa, in most instances, is produced by chronic trauma, either physical or chemical, or has origin in a premalignant lesion as leukoplakia, papilloma or chronic ulcer. Any chronic ulcer in the mouth should have adequate biopsy to determine the presence of malignancy. Small cancers located on the soft or hard palate, upper or lower gingiva or buccal mucosa may be eradicated by roentgen therapy through an intraoral cone or by electrosurgical excision with good results. Larger cancers which invade the superior maxilla, mandible, antrum, or deep structures of the cheek require more intensive irradiation therapy followed by more radical surgical excision.

The implantation of radium element needles in intraoral cancer is completely obsolete. This modality of therapy seldom controls the malignancy, invites infection, fibrosis, and scarring, results in a painful indolent ulcer and possible radioosteonecrosis, osteomyelitis and ankylosis of the jaw.

If there is clinical evidence lymph nodes in the drainage area are involved, radical neck dissection should be carried out following control of the primary lesion. Patients with extensive malignancies may be cured with intensive roentgen therapy followed by electrosurgical removal of the tumor. The defect is covered with a pedicle graft. Modern prostheses which give good functional and cosmetic results are utilized when it is necessary to resect the superior maxilla, soft and hard palate, or mandible.

The illustrations are from Ward and Hendricks: *Tumors of the Head and Neck*, Williams and Wilkins Company, with kind permission.

REFERENCES

1. Ackerman, A. J.: Protective Appliances for the Oral Cavity and Face During Radiation Therapy. Chap. 28, Treatment of Cancer and Allied Diseases by Pack, G. T., and Livingston, E. M. Paul B. Hoeber, N. Y., 1940.
2. Archer, W. H., and Morris, R. D.: Survey of Oral Carcinoma at Columbia-Presbyterian Medical Center, *Amer Jour. Orthodont.* 32:338, 1946.
3. Berven, E. G. E.: Radiological Treatment of Malignant Tumours of the Oral Cavity and Pharynx, *Acta Radiol.* 18:16, 1937.
4. Blair, V. P., Brown, J. B., and Womack, N. A.: Cancer in and about the Mouth; A Study of 211 Cases, *Ann. Surg.* 88:705, 1928.
5. Blair, V. P., Brown, J. B., and Womack, N. A., and Byars, L. T.: Our Responsibility Toward Oral Cancer, *Ann. Surg.* 106:568, 1937.
6. Davis, G. G.: Buoy Cheek Cancer, *J. A. M. A.* 64:711, 1915.
7. Erick, J. B.: Surgical Excision of Small Cheek Cancers, *Surg. Clin. N. A.* pp. 1017 (Aug.) 1941.
8. Esser, J. F.: Studies in Plastic Surgery of the Face, *Amer. Jour. Surg.* 65:297, 1917.
9. Figi, F. A.: Malignant Diseases of the Mouth, *Jour. Amer. Dental Assoc.* 26:1979, 1939.
10. Hendrick, J. W., Ward, G. E., and Lacy, M. M.: Salivary Tissue Neoplasms, *Amer. Jour. Surg.* 81:373, 1951.
11. Jackson, H. S., and New, G. B.: Treatment of Intraoral Cancer, *Surg., Gynec. and Obst.* 91:232, 1950.
12. Kelly, H. A., and Ward, G. E.: Electrosurgery, W. B. Saunders, Co., Philadelphia, 1919.
13. Lund, C. C.: Epidermoid Cancer of the Buccal Mucosa, *Surg., Gynec. and Obst.* 66:810, 1938.
14. Martin, H. E.: Peroral X-radiation in the Treatment of Intraoral Cancer, *Radiol.* 28:527, 1937.
15. Martin, H. E.: Tumors of the Palate (Benign and Malignant), *Arch. Surg.* 44:599, 1942.
16. Martin, H. E.: Personal Communication.
17. Martin, H. E., and Koop, C. E.: The Precancerous Mouth Lesions of Avitaminosis "B," *Amer. Jour. Surg.* 57:195, 1942.
18. Martin, H. E., and Pflueger O. H.: Cancer of the Cheek (Buccal Mucosa), *Arch Surg.* 30:731, 1935.
19. MacFee, W. F.: Resection of the Upper Jaw for Carcinoma, *Amer. Jour. Surg.* 30:21, 1935.
20. MacFee, W. F.: Full Thickness Defects of the Cheek Involving the Angle of the Mouth, *Surg., Gynec. and Obst.* 76:100, 1943.
21. New, G. B., and Cabot, C. M.: The Curability of Malignant Tumors of the Upper Jaw, *Surg., Gynec. and Obst.* 60:971, 1935.
22. New, G. B., and Cabot, C. M.: Malignant Disease of the Mouth and Accessory Structures, *Amer. Jour. Surg.* 30:46, 1935.
23. New, G. B., Cabot, C. M., and Holberg, O. E.: End Results of Treatment of malignant Tumors of the Palate, *Surg., Gynec. and Obst.* 73:520, 1941.
24. Polya, E.: Technique of Operations for Carcinoma of Buccal Mucous Membrane, *Surg., Gynec. and Obst.* 43:343, 1926.
25. Pyott, J. E., Bruder, V. F. J., Manion, W. J., and Ward, G. E.: Leaded Resinous Applicator for Intraoral and Extraoral Radium Therapy, *Amer. Jour. Roentgen. and Rad. Therapy* 47:613, 1942.

26. Richards, G. E.: Radiation Therapy of Carcinoma of the Buccal Mucosa (Cheek), Chap. 16, vol. 1, Treatment of Cancer and Allied Diseases by Pack, G. T., and Livingston, E. M. Paul B. Hoeber, N. Y., 1940.
27. Simmons, C. C.: Cancer of the Buccal Mucosa, *Ann. Surg.* 92:681, 1930.
28. Taylor, G. W.: Cancer of the Buccal Mucosa, *Surg., Gynec. and Obst.* 58:914, 1934.
29. Thoma, K. H.: Oral Pathology, 2nd Ed., C. V. Mosby Co., St. Louis, 1944.
30. Vital Statistics of the United States—Special Reports, vol. 9, No. 25.
31. Ward, G. E.: A Conservative Operation for the Cure of Ranula by Endothermy, *Med. Rev.* 31:587, 1925.
32. Ward, G. E., and Duff, A. M., Jr.: Tumors of the Tongue, *Cyclopedia of Med., Surg. and Specialties*, 1940, F. A. Davis Co., Philadelphia.
33. Ward, G. E., and Edgerton, M. E.: Recent Improvements in Resection of the Maxilla, *Amer. Jour. Surg.* 80:909, 1950.
34. Ward, G. E., and Hendrick, J. W.: Tumors of the Head and Neck, Williams and Wilkins Co., Baltimore, 1950.
35. Ward, G. E., and Hendrick, J. W.: End Results in Treatment of Cancer of the Lip, *Surgery* 27:321, 1950.
36. Ward, G. E., and Hendrick, J. W.: Tumors of the Jaws, *Jour. Internat. Coll. Surg.* 15:443, 1951.
37. Watson, W. L.: Adenocarcinoma of the Oral Cavity, *Amer. Jour. Roent.* 34:53, 1935.
38. Whitmore, E. R.: The Nature of Metaplasia and of Malignant "Degeneration," *Boletin de la Liga contra el cancer* 13:263, 1938.

The Scientific Papers of the American Broncho-Esophagological Association

XC

TRAUMATIC RUPTURE OF THE LOWER TRACHEA WITH STENOSIS

A CASE REPORT

G. ARNOLD HENRY, M.D.

TORONTO, CANADA

In this presentation, the lower trachea will consist essentially of the thoracic portion of that tube. More exactly, it will be that portion of the trachea lying below the lowest tracheotomy site. It now becomes evident that the diagnosis and treatment of traumatic rupture in this area will be quite different from that in its upper portion.

Traumatic rupture of the lower trachea is not common, if we may judge by reported cases. It occurs usually following a crushing chest injury, but may be caused by a sudden tracheal strain, or during bronchoscopy, as reported by Andrews and McMahon.¹

Zeuch,² in a survey of the literature to 1922, reported 53 cases of subcutaneous rupture of the trachea. Eight of these appeared to occur in the lower trachea. Other than that three lived, details were not available.

Nixon,³ in 1925, reported a case somewhat similar to the one today, caused by sudden tracheal strain. The exact site of injury was not observed, and the patient recovered following tracheotomy.

More recently, Holinger, Johnston and Besenger,⁴ in a paper on Benign Stenosis of the Trachea, report two cases of fractures of the intrathoracic portion of the trachea due to automobile accident. The case described suffered a stenosis which was discovered at a later date when the patient, after laryngectomy for carcinoma of the larynx, was being intubated.

REPORT OF A CASE

A young male industrial worker, aged 17, was admitted to St. Michael's Hospital, Toronto, on April 26, 1947. An hour or two earlier, his left hand, arm and shoulder were caught and drawn in between two huge paper rollers. He was released, unconscious, after he had been pulled up into the air.

On admission, the patient was shocked, dyspneic, and exhibited interstitial emphysema over his neck and left upper chest. Indrawing occurred in the lower intercostal spaces on inspiration. Occasionally, he coughed up a little blood. The left vocal cord did not move. Clinically and radiologically, there was no evidence of bony fracture of the thorax. A pneumothorax was noted in the upper left chest-area.

Over the next two weeks, the patient made an almost complete recovery. Then, on May 11th—fifteen days following the accident—the patient became dyspneic. Both sides of the chest were hyper-resonant. Feeling that this might be caused by a tension pneumothorax, an attempt was made to withdraw air. Some air was withdrawn with temporary relief.

In a sudden turn for the worse, the patient, even with oxygen by mask, was conscious only at times. Inspiratory indrawing was present bilaterally in the upper intercostal spaces. X-rays now showed a gross bilateral emphysema of the type seen in early foreign body reaction, where air is trapped in the lung in the inspiratory phase. It was felt that the obstruction must be tracheal.

The patient was taken to the operating room, the oxygen mask removed, and a bronchoscope passed. The trachea was found to be torn, starting about one and one-half inches above the carina. Around the tear was a collar of granulation tissue, which was causing the obstruction. The 'scope was passed through this and the patient regained consciousness.

With the bronchoscope in situ, the lowest possible tracheotomy opening was made and a rubber intratracheal tube was inserted. We were troubled by thick secretions and crusting, which necessitated frequent suctioning, tube changing, and occasional bronchoscopic removal. Long tracheotomy tubes of the walking-stick type were obtained. In order to pass the injured area, they frequently rode the carina, in turn causing obstruction by this irritation. (Slides)

About May 25—a month after the accident—the injured trachea showed signs of stenosis, and bits of cartilage from three or four tra-

cheal rings began to extrude themselves into the lumen, requiring bronchoscopic removal.

About this time, we were able to have made more exact plastic and metal tubes which made our task easier. Dilatation of the stenosed area was carried out on alternate days with metal dilators through the tracheotomy opening.

In early September, four months after the injury, the tracheal wall was well-lined with mucous membrane and the stenosis had disappeared. There was, however, an area about one inch long, about one-half inch above the carina, where, through loss of cartilaginous support, the trachea tended to collapse on inspiration. The patient learned to change his own tube and was discharged.

Examination 10 days ago showed a fairly normal tracheal lumen as to size and appearance. There is considerable stiffening of the tracheal wall. However, it is evident that it is not firm enough for decannulation.

During all this time, the patient has had nearly 40 bronchoscopic procedures. He is well, has been at work for four years at his same old job, and plays a good game of tennis.

We are aware of the excellent experimental work by Taffel,⁵ Daniel, Taliaferro and Schaffarzich,⁶ and Clagget, Grindlay and Moersch⁷ in the closure of tracheal defects in dogs. We hope that something better may be done eventually for this man.

CONCLUSIONS

Traumatic rupture of the lower trachea may occur as the result of a severe chest injury or, indirectly, because of a sudden tracheal strain.

Evidence of such an injury will be manifested by sudden shock, dyspnea, interstitial emphysema over the neck and chest, and the expectoration of blood.

Localization of the site of the injury in the respiratory tree may be difficult because of pneumothorax and other complicating injuries. Severe tracheal obstruction, if it occurs, may be immediate or some time after the accident. Tracheal obstruction will become apparent when the clinical and radiological findings show a symmetrical disturbance of the mechanics of the chest. This will be evidenced by bilateral indrawing in the intercostal spaces and bilateral obstructive emphysema.

Bronchoscopic examination should be made early to determine the site of the injury. This will be followed, in most instances, by a low tracheotomy and the use of suitable tubes to maintain an airway.

Stenosis may be kept in check by the use of as large a tracheotomy tube as possible, bronchoscopic dilatations, and the passage of tracheal dilators through the tracheotomy wound.

Where the loss of tracheal rings occurs, the treatment is still obscure. Since it would appear that the remaining fibrous channel is unlikely to form a safe airway, we must hope that experimental surgery will, in the near future, offer a reasonable chance in such an injury.

REFERENCES

1. Andrews, A. H., Jr., and McMahon, R. L.: *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 59:726, 1950.
2. Zeuch, L. H.: *Illinois M. J.* p. 451 (June) 1922.
3. Nixon, J. W.: *J.A.M.A.* 85:1221, 1925.
4. Holinger, P. H., Johnston, K. C., and Besenger, C. E.: *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 59:837, 1950.
5. Taffel, M.: *Surgery* 8:56, 1940.
6. Daniel, R. A., Jr., Taliaferro, R. M., and Schaffarzich, W. R.: *Diseases of the Chest* 17:426, 1950.
7. Clagett, O. T., Grindley, J. H., and Moersch, H. J.: *Arch. Surg.* 57:253, 1948.

DISCUSSIONS

DR. GEORGE S. McREYNOLDS (Galveston, Texas): I think this paper is particularly apropos, following the symposium this morning, on the relationship of endoscopy to thoracic surgery. It requires complete co-operation between the endoscopist and thoracic surgeon to evaluate the condition in these cases.

We recently had a case, which we intended to report in more detail, of a young lady in an automobile accident. She received an injury to her chest from the steering wheel without any particular external evidence of it. There were no symptoms for a week. Then she developed traumatic necrosis between the esophagus and the trachea and a fistula. Every time she attempted to drink water, terrific coughing would ensue.

There was no roentgenographic evidence of disease of the chest. The lung fields were clear. The mediastinum showed no evidence of involvement and no widening. There was no febrile reaction nor any leucocytic response. It was only through suspicion and the help of the patient that we had any concrete evidence that there was an injury to the trachea and esophagus.

DR. WALTER HUGH MALONEY (Philadelphia, Pa.): The treatment of a rupture of a trachea sometimes becomes rather an acute emergency procedure, and

is usually concomitant with the mediastinal drainage and the tracheotomy. The immediate relief of dyspnea is usually the acute problem.

I would like to raise a point of discussion both with the author and with the other members as to the sequence of events in the treatment of rupture of the trachea. Granted that it is important to localize the point of rupture, the method of doing it seems to me to be important, too.

May I quote a case: A six year old boy was admitted to the hospital one hour after he had fallen from his bicycle. His mother said that he had had an increase in the size of his head. It was obvious that he had a subcutaneous emphysema which was rapidly increasing. There was no sign of any external trauma. The child was alert, clear voiced, cooperative; the only physical finding was subcutaneous emphysema.

We felt that he had a rupture of the trachea. We did not know whether it was a high or a low tracheal rupture, or a bronchial rupture.

Since the child did have a rapidly progressing emphysema, I elected to do an endoscopic examination in an attempt to find the exact point of rupture. By the time I had finished the bronchoscopy, operation was no longer an elective procedure. The child was extremely dyspneic, the subcutaneous emphysema now extended down to his toes, and the mediastinal drainage and tracheotomy were performed as emergency procedures.

On opening the trachea, I found a tremendous rupture of the membranous posterior portion from the 2nd to the 6th tracheal cartilage. In retrospect, I wonder if one is justified in an immediate bronchoscopy or if, instead, tracheotomy and mediastinal drainage should be done first to avoid the increase in pressure caused by closure of the glottis on crying and straining, which in turn increases the mediastinal emphysema and tracheobronchial compression.

I think it is a rather rare to have a rupture of a trachea with no physical findings other than subcutaneous emphysema.

DR. LOUIS H. CLERF (Philadelphia, Pa.): A number of years ago, I presented before this Association, two patients who had progressive subcutaneous emphysema involving the upper extremity, head and neck. Both of these patients had a peanut in the bronchus. The diagnosis was made on the basis of radiographic study because there was so much air in the tissues over the chest that the internist could not carry out a satisfactory physical examination.

I have observed such a case as has been described here, a traumatic lesion of the trachea occurring during an automobile accident in the wife of a physician. She was practically unconscious, also dyspneic. There was hemoptysis and emphysema of the neck and head.

Roentgen study revealed mediastinal emphysema. Tracheotomy was done for relief of dyspnea but it was not adequate. A bronchoscopy was done and a laceration of the thoracic portion of the trachea, beginning at the suprasternal notch, was discovered.

There had been no gross evidence of any trauma to the chest. It was difficult to evaluate this because of the extensive damage and the tracheal cannula previously introduced was believed to be inadequate because it did not get beyond the point where the rupture or the laceration of the trachea appeared. So a cane-shaped cannula was introduced, and that did relieve the situation.

The question arose what to do and the thoracic surgeon, after consultation, decided he ought to do something more than merely insert a cannula. A thoracotomy through the sternum was done and a considerable portion of the trachea was found to be lacerated.

A tantalum tube 7 cm in length and 11 mm in diameter was inserted and secured with steel wire.

The anchoring wire was removed two months later and within a few hours the tantalum tube descended into the trachea, the lower end entering the right bronchus. A bronchoscopy was done but the trachea could not be traversed, where the tube had been, due to stenosis.

What had happened was that the moment we removed the steel suture, the tube slowly moved down and the cicatrized trachea began to narrow to a point where it was almost impossible to maintain respiration.

The surgeon split open the sternum again and found an enormous cicatricial mass. He had great difficulty in finding the tracheal lumen. A tracheal cannula was inserted.

The cicatricial mass which enveloped practically the entire thoracic trachea was opened, the tantalum tube was removed and after removing considerable cicatricial tissue, a polyethylene tube was substituted.

I have seen the patient at intervals since, and the tube still is in situ and it hasn't moved either up or down. We are able to traverse it with a six millimeter bronchoscope. She is having a little difficulty with secretions.

We are now wondering what to do. If anybody has a suggestion, I would be glad to take it home with me.

DR. EDWARD B. BENEDICT (Boston, Mass.): About a year ago, Dr. J. Gordon Scannell reported a case of ruptured bronchus in the *Annals of Surgery*.^{*} He made the diagnosis immediately and no preoperative bronchoscopy was done. He did a thoracotomy and an immediate suture at once with complete recovery of the patient.

I wonder whether the bronchoscopist should interfere in cases like this or should it be left entirely to the thoracic surgeon?

DR. G. ARNOLD HENRY (Toronto, Canada): It has been a great deal of pleasure for me, as a guest, to appear on this program today.

Dr. Maloney mentioned something about the sequence of events, and I don't know that I can answer, because I think each case must be different from the rest. The amount of dyspnea and the amount of shock at the beginning must vary a great deal with the amount of pneumothorax that is present at the time and the amount of bleeding that is present with the rupture of the trachea.

In this case, what I first saw was fairly marked dyspnea, but less than he had two weeks later when organization of the granulation tissue occurred.

^{*}Rupture of the Bronchus Following Closed Injury to the Chest, Report of a Case Treated by Immediate Thoracotomy and Repair, *Ann. Surg.* 133:127-130 (Jan.) 1951.

STENOSIS OF THE ESOPHAGUS IN BENIGN MUCOUS
MEMBRANE PEMPHIGUS

EDWARD B. BENEDICT, M.D.

AND

WALTER F. LEVER, M.D. (By Invitation)

BOSTON, MASS.

Although it is well known that esophageal obstruction may be caused by various types of inflammatory processes, chemical poisons, tumors, webs, and neuromuscular difficulties,^{1, 2} the association of dysphagia with benign mucous membrane pemphigus has received very little recognition. Benign mucous membrane pemphigus, also called pemphigus conjunctivae, may affect the mucous membrane of the esophagus and cause obstruction of the lumen. This fact is not mentioned in dermatologic textbooks and is only briefly referred to in one book on esophageal disease.³ It therefore seems indicated to review the literature on this subject and to report the findings in four patients with benign mucous membrane pemphigus in whom involvement of the esophagus was present. First, however, the disease benign mucous membrane pemphigus will be briefly defined.

DEFINITION OF BENIGN MUCOUS MEMBRANE PEMPHIGUS

Benign mucous membrane pemphigus is a disease entity which is not related to pemphigus vulgaris, pemphigus foliaceus or pemphigus vegetans. It differs from these three forms of true pemphigus in its clinical appearance and in its course as well as in the histologic structure of the bulla.

Clinical Appearance: Benign mucous membrane pemphigus is characterized by the presence of bullous lesions and erosions on the mucous membranes. In about half of the cases lesions are found also on the skin. Scarring invariably develops on the mucous membranes and occasionally on the skin. This tendency to scarring is peculiar to benign mucous membrane pemphigus and does not occur in the other forms of pemphigus.

From the Departments of Surgery and Dermatology, Harvard Medical School and Massachusetts General Hospital.

The conjunctivae are the most frequently affected site and occasionally represent the only area of involvement; on the other hand, they may be free from lesions. The conjunctival lesions invariably lead to scarring and frequently to destruction of the cornea and thus cause blindness. Since the conjunctival lesions are in the majority of patients, the most distressing feature, ophthalmologists were the first to describe the disease.⁴ They conferred upon it the name pemphigus conjunctivae.

Of other mucous membranes the oral mucosa is next in frequency of involvement. The nasal mucosa, larynx, esophagus, glans penis, and the vulvar and anal mucosa are occasionally involved.

The cutaneous lesions are of two types. They consist either of discrete bullae which heal within a short period of time or of erythematous areas in which bullae recur over a long period of time until gradually an area of smooth atrophy results.⁵

Course. Benign mucous membrane pemphigus follows an extremely chronic, benign course. In contrast to pemphigus vulgaris, pemphigus foliaceus and pemphigus vegetans the general health is not affected and death from the disease is rare. Occasionally, death may result from respiratory distress with ultimate bronchopneumonia.

Histopathology. The bullae of benign mucous membrane pemphigus are subepidermal in location, whether they occur on the skin or on the mucous membranes.⁶ The epidermis overlying the bullae shows no primary degenerative changes. This is in contrast to pemphigus vulgaris, pemphigus foliaceus and pemphigus vegetans. In the latter three diseases degenerative changes in epidermal cells are the primary alteration leading to a loss of the intercellular bridges and thus to separation of the epidermal cells from one another. This process referred to as acantholysis results in bullae in intra-epidermal rather than subepidermal location.

REVIEW OF LITERATURE

The first case of benign mucous membrane pemphigus with involvement of the esophagus was reported in 1910 by Adam.⁷ This case concerned a 55-year-old woman who complained of inability to get down any liquids or solid food. Examination with a bougie revealed one obstruction in the pharynx right below the larynx and another more severe stenosis somewhat deeper, 23 cm from the teeth. Passage of the bougie beyond this point was impossible. The following day the patient had a high fever. She died on the third day. Autopsy showed two perforations in the upper esophagus which had

resulted in mediastinitis and empyema. The upper portion of the esophagus and the entrance to the larynx showed severe scarring. The writer believed that the stricture had been produced by pemphigus blisters and by edema of the mucosa.

In 1911 Thost⁸ reported a case of benign mucous membrane pemphigus in which involvement of the esophagus had begun in the tenth year of the disease. The patient complained that food tended to get stuck in his throat. The esophagus showed at the entrance large white membranes. Six years later Thost⁹ described the autopsy findings in this case. The disease had started at the age of 50 with nasal symptoms. Dysphagia had begun at the age of 60. Two years later a bolus got stuck in the esophagus and had to be pushed down with a bougie. A similar episode occurred a few months later. Thereafter the patient was able to eat only finely-ground food. He died of pneumonia at the age of 82. Autopsy disclosed diffuse shrinking and folding of the esophageal mucosa. The folding was longitudinal. Just as on the conjunctivae, there were webs caused by the growing together of parts that normally touch each other. In the upper esophagus a firm, thick fold was present which had caused a small, accessory lumen, the diameter of a pencil, to be separated from the greatly narrowed main lumen. There were no blisters in the esophagus. Histologic examination of the esophagus revealed the epithelium to be well preserved. The submucosa was thickened and fibrotic with a considerable infiltrate of lymphocytes and plasma cells.

In 1931 Imperatori¹⁰ reported the case of a 68 year old man whose chief complaint was difficulty in swallowing of ten months' duration. He had had benign mucous membrane pemphigus of the mouth, pharynx, conjunctivae, and left side of his face for a period of 13 years. A roentgenogram showed a stricture at the sixth cervical vertebra that greatly reduced the caliber of the esophagus so that it only permitted the passage of a three millimeter stream of barium. Esophagoscopy revealed a stricture at the cricopharyngeus through which a number 14 (French) bougie was passed with some difficulty. Later a number 24 (French) was introduced and left in situ for 10 minutes. The mucous membrane looked as if cobwebs were scattered over it. The day after divulsion of the stricture the patient was able to swallow without difficulty and continued to do so up to the time of the last report, six months later.

In 1933 Baumann¹¹ reported the case of a 61 year old woman who had been admitted to the hospital in an extreme state of malnutrition and dehydration, supposedly suffering from cancer of the esophagus. Four years before admission her eyelids had gradually

become adherent and a diagnosis of ocular pemphigus had been made. About six months before entry she had experienced difficulty in swallowing solid food which had become progressively worse. At the time of entry she could take only small quantities of liquids. Laryngoscopy and esophagoscopy showed the following picture: "The epiglottis was deeply congested, infiltrated and showed a few tiny blebs on the upper area. About two centimeters below the cricopharyngeus there was a tight stricture of the esophagus. The stricture seemed to be about three centimeters in length. The membrane was not ulcerated and the walls did not seem to be infiltrated. The stenosis was dilated and a feeding tube was introduced." The feeding tube was left in situ for five days. When it was removed, radiologic studies no longer revealed any evidence of a stricture of the esophagus. Following the dilatation the patient was able to take strained or ground foods. One year later she had gained 35 pounds in weight and was doing well.

In 1951, Greither¹² reported a case of "chronic aphthosis" which strikingly resembles our Case I and which we are inclined to regard as an instance of benign mucous membrane pemphigus. At 29 years of age oral lesions appeared. At the age of 41 the patient began to have dysphagia which at times forced her to take liquid foods exclusively. On examination at the age of 42 esophagoscopy revealed edema of the esophageal mucosa with whitish shreds of detached epithelium. The oral mucosa showed extensive erosions. The small labia of the vulva were firmly adherent to one another in their upper portion and the lumen of the vagina was considerably narrowed. The dysphagia was treated by repeated bouginage with great improvement each time. The oral erosions gradually extended to the lips and corners of the mouth. At the age of 46 an indurated area first appeared on the lower lip and gradually increased in size. Histologic examination revealed this lesion to be a squamous cell carcinoma.

REPORT OF CASES

CASE 1.—E. M. M. MGH Nr. U-1722747. A 71-year-old woman¹³ was first admitted to the Massachusetts General Hospital on January 9, 1951 complaining of swelling and nodular induration of the lower lip, dry eyes, soreness of the mouth, and difficulty in swallowing. The present illness had begun about 30 years ago with the development of painful lesions in the mouth. Ever since oral lesions have continuously appeared in the mouth. They gradually extended to the vermilion border of the lips. About 20 years ago the mucous membranes of the eyes and of the vulva became involved. For about the same length of time, the patient has had difficulty in swallowing

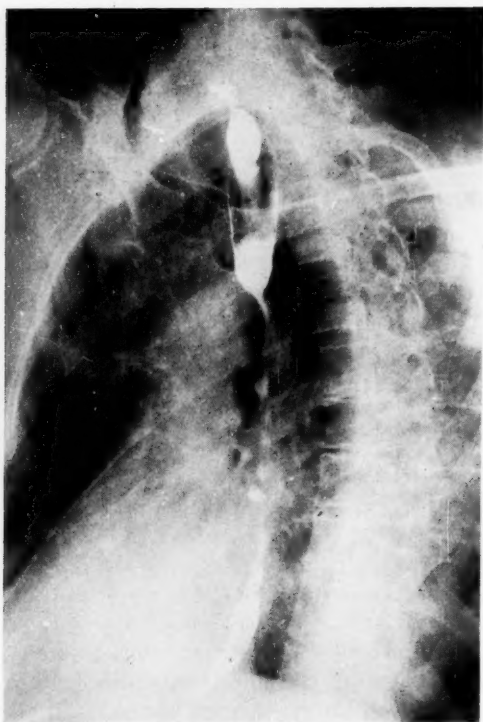


Fig. 1, Case 1.—X-ray appearance showing multiple strictures of the esophagus beginning at the episternal notch.

food. An esophagoscopy was performed five years ago because a pea had become lodged in her throat. At that time it had been noted that there were some webs in the esophagus. Since then the patient has had periodic bouginage about once every two or three months because of her dysphagia. X-ray examination (Fig. 1) one week prior to admission demonstrated multiple strictures of the esophagus beginning at the episternal notch.

On admission the patient showed conjunctival adhesions of both eyes and extensive oral erosions. The lower lip showed diffuse nodular induration. The vaginal introitus was greatly narrowed due to atrophic scarring. Biopsy of a nodular lesion on the lower lip revealed squamous cell carcinoma, grade I.

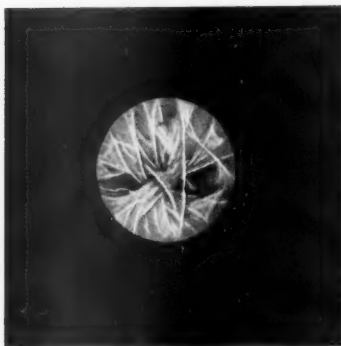


Fig. 2, Case 1.—Drawing at time of esophagoscopy. Benign mucous membrane pemphigus showing vertical and oblique membranous obstruction of the esophagus.

Esophagoscopy on January 12, 1951 gave the following findings:

The esophagoscope passed readily into the upper esophagus. The esophagus appeared normal down to a point about 5 cm below the cricopharyngeus where there was a web-like membranous obstruction with a 3 mm circular opening in the middle of it. This was quite readily divulsed with bougies sizes 12 to 20 inclusive; after which it was possible to see about 3 cm. below this level where there were vertical membranous structures (Fig. 2) apparently dividing the esophagus into two parts with the main lumen appearing to go to the right and a small opening to the left. It was not thought advisable to perform a biopsy or to introduce the esophagoscope any farther.

Conclusions: The finding of web-like structures is consistent with a diagnosis of benign mucous membrane pemphigus of the esophagus. The webs appear similar to those seen in this patient's conjunctivae.

Radiologic examination (Fig. 3) on January 19, 1951 showed some improvement following esophagoscopy. There was, however, a constant, conical narrowing of the esophagus at the level of the aortic arch. Another constant narrowing was noted about 11 cm. above the diaphragm. This narrowing presented the appearance of a shelf deformity and looked more like a tumor than a benign process. Despite the clinical and esophagoscopic findings of pemphigus the

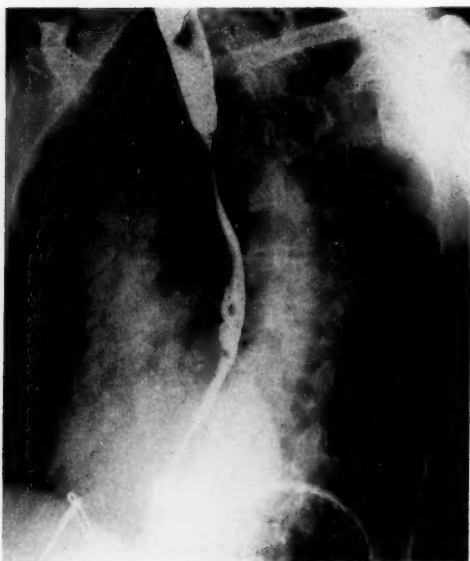


Fig. 3, Case 1.—X-ray appearance of esophageal lesions, showing some improvement after bouginage.

radiologist still wondered if this could not be carcinoma of the esophagus.

In order to accomplish further dilatation of the esophagus the patient was instructed to swallow a thread and a number 19 bougie was passed using the thread as a guide. Two days later a number 25 bougie passed readily. Three weeks later the patient stated that she could swallow food much better than formerly. Bougies sizes 29, 31 and 33 were passed. Because of the suspicious shelf-like formation visualized by radiologic examination esophagoscopy was repeated on February 9, 1951.

The esophagoscope passed readily to a point about 6 cm below the cricopharyngeus where the lumen was narrowed in a circular manner to a diameter of about 5 mm with diffuse redness and edema. Bougies sizes 20 and 22 passed readily, after which it was possible to divulse the narrowing still further by passing the entire esophagoscope. The esophagus then appeared essentially normal throughout its mid-portion, but at a point 11 cm above the diaphragm there was a shelf-like membranous web obstructing about two-thirds of the

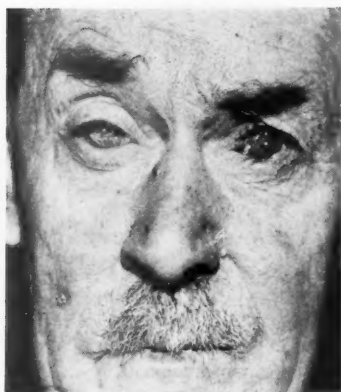


Fig. 4, Case 2.—Benign mucous membrane pemphigus involving conjunctiva cornea and face.

lumen. Bougies sizes 20 and 22 passed readily but the esophagoscope would not pass. A satisfactory biopsy was obtained of this web-like structure with the cutting forceps. The lesion did not suggest carcinoma.

Conclusions: The findings were those of multiple web-like strictures of the esophagus consistent with benign mucous membrane pemphigus.

The biopsy showed nonspecific chronic inflammation. A positive diagnosis of mucous membrane pemphigus could not be made from the biopsy.

The carcinoma of the lower lip was at first treated by x-ray and later was widely resected.

CASE 2.—A. R., MGH Nr. U-672366, a 71-year-old man first entered the hospital in August 1949 because of photophobia with diminishing vision and moderately itching lesions of the skin of one year's duration.

On physical examination both eyes showed numerous conjunctival adhesions. The cornea of the right eye was almost completely obliterated with adhesions and there was a dense opacity overlying the pupil (Fig. 4). Cutaneous lesions were present on the scalp, face, back, chest, abdomen, and arms. They were circumscribed. Some showed flaccid vesicles, others were erosions and still others were



Fig. 5, Case 2.—Drawing at time of esophagoscopy. Benign mucous membrane pemphigus showing transverse membranous web.

crusted. A few showed no more activity but atrophic scarring. A diagnosis of benign mucous membrane pemphigus was made.

The lesions of the eyes progressed gradually and by February 1951 the patient was totally blind due to complete opacity of both corneas. Oral lesions consisting of vesicles and erosions were first noted in July 1950. A biopsy of a lesion on the soft palate performed in February 1951 showed a subepidermal bulla with no acantholysis and therefore was interpreted as consistent with benign mucous membrane pemphigus. In April 1951 the patient's mouth and throat became rather sore so that eating was difficult. In June 1951, food seemed to get stuck at the level of the sternal notch. Radiologic examination on June 19, 1951 showed two constricted areas in the upper cervical esophagus just below the hypopharynx. They were thought to be either redundant mucosal folds, webs, or strictures of the esophagus.

Esophagoscopy two days later revealed the following:

The esophagus appeared normal down to a point only 5 to 6 cm below the cricopharyngeus where there was a membranous web stretching across the lumen of the esophagus horizontally with an opening only 2 to 3 mm in diameter (Fig. 5). The opening was circular and was partially divulsed with a bronchoscopic sponge, after which the entire esophagoscope passed readily into the distal esophagus and down to the cardiac orifice, all of which appeared normal. No web was seen at the level of the cricopharyngeus and the web seen by esophagoscopy seemed to be somewhat lower than that visible on the x-ray film. Possibly there was more than one web.

Conclusions: The findings were consistent with a web of the esophagus, readily divulsed.

Following this procedure the patient was able to swallow food without difficulty. Intake, however, was limited to finely ground food because of his sore mouth. Radiologic examination a month after the esophagoscopy demonstrated an essentially normal appearing esophagus. When seen in January 1952 the patient had no dysphagia. His weight and appetite were good.

Dysphagia recurred, however, in March 1952. Esophagoscopy was therefore repeated April 4, 1952. The findings were much the same as described ten months previously. There was again a membranous web-like formation with a circular opening in the middle. There was considerable reddening and some edema. Bougies sizes 14 to 26 passed readily divulsing the web. However, the esophagoscope would not pass beyond that level.

Comment: The fact that the esophagoscope passed the web on the first occasion and failed to pass it at the time of the second esophagoscopy would tend to indicate a progression of the esophageal stenosis.

CASE 3.—A. B., MGH No. U-718967, a 69-year-old woman, was admitted to the hospital in November 1950 because of sores in her mouth and difficulty in swallowing of eight months' duration.

Physical examination revealed extensive oral erosions extending to the vermilion border of the lips. Several adhesions were present between the gums and the buccal mucosa. The uvula was obliterated. The pharynx showed several eroded areas. No conjunctival lesions were noted. Barium swallow failed to demonstrate any evidence of esophageal stricture or disorder in the swallowing function.

Esophagoscopy, however, on January 31, 1951, six weeks after the radiologic examination, gave the following findings:

The hypopharynx appeared diffusely inflamed with a somewhat granular appearance. The mucosa bled very easily. There were several erosions with loose pieces of membranous tissue just above the cricopharyngeus.

The inflammation extended to the region of the cricopharyngeus where a considerable delay was encountered to the passage of the esophagoscope. As it finally passed, it apparently divulsed a web-like structure right at the cricopharyngeus. The instrument was then passed all the way through a normal appearing esophagus to the cardiac orifice and through it into the upper part of the stomach, which also appeared normal.

Conclusions: The findings were consistent with a web at the cricopharyngeus, probably on the basis of benign mucous membrane pemphigus.

Two weeks later the swallowing was much improved and the patient was able to eat everything.

On March 9, 1952, following her lunch, the patient experienced sudden inability of swallowing her saliva. Esophagoscopy examination revealed a bolus of meat in the esophagus just below the cricopharyngeus. Upon its removal a small stricture was visualized at that site. A bougie number 36 could be passed with ease beyond the stricture. No erosions or any other strictures were noted in the esophagus. Since this episode the patient has had no further difficulty in swallowing.

CASE 4.—W. E. E., MGH Nr. U-119970, a 67-year-old man entered the hospital on July 15, 1929 with a history of soreness in the throat for a year and a half, and soreness in the mouth for one year. For the past six months he had noted difficulty in swallowing food unless it was very finely chewed.

On physical examination the mouth showed diffuse redness around the gums and on the palate. Two small erosions were present on the palate. The left eye was injected and there were several adhesions between the palpebral and bulbar conjunctivae.

Nine years later, on April 1, 1938, the patient returned to the hospital because of increasing difficulty in swallowing. Radiologic examination demonstrated a web in the upper third of the esophagus. For purpose of dilating the esophagus bougies were passed at varying intervals during the next six years. A number 28 (French) was used at first but later the esophagus tolerated a bougie as large as number 37.

In 1944 the patient stated that he no longer had any difficulty in swallowing except meat which still had to be ground up finely. Unfortunately, esophagoscopy was never performed. He was last seen in November 1944 at which time he was 82 years old. Attempts at follow-up have been unsuccessful.

COMMENT

The esophageal lesions in benign mucous membrane pemphigus consist of diffuse inflammation of the mucosa and the submucosa. Bullae have so far not been described in the esophagus. This may be due to the fact that examination of the esophagus has been carried

out only in cases of long standing in which bulla formation has already been superseded by scarring. The scarring in the esophagus leads to soft adhesions which by roentgenologic examination and by inspection with the esophagoscope have the appearance of webs. These adhesions are thin and soft at first but, in the course of time, they become firmer. They may differ from other esophageal webs in having the appearance of fine thread-like adhesions resembling cobwebs running vertically, obliquely or transversely.

It is believed that recognition of esophageal involvement in benign mucous membrane pemphigus is important, because the soft adhesions, as present in the early stage, can easily be divulsed, whereas the adhesions in their late stage may resist treatment by bouginage. Any patient with benign mucous membrane pemphigus complaining of dysphagia should therefore be given a roentgenologic examination of the esophagus. Since webs may not be demonstrable by x-ray, esophagoscopy should be performed, regardless of the x-ray findings.

The process taking place in the esophagus is comparable with that affecting the conjunctivae. The conjunctivae also show at first diffuse inflammation with occasional bulla formation. This gradually leads to the formation of adhesions which extend between the tarsal and bulbar conjunctivae of each lid as well as between the conjunctivae of the lower and upper lids. These conjunctival adhesions are soft and pliable at first but with time they become fibrous and rigid.

SUMMARY

Benign mucous membrane pemphigus differs from pemphigus vulgaris, pemphigus foliaceus and pemphigus vegetans by its predilection for the mucous membranes, the tendency of the lesions to produce scarring, its chronic, benign course and the subepidermal location of the bullae.

Of the various mucous membranes the conjunctivae and oral mucosa are most commonly affected. The esophagus shows occasional involvement resulting in web-like adhesions which narrow the lumen of the esophagus.

Early recognition of the esophageal lesions is important because in their early stage the adhesions can be easily divulsed.

REFERENCES

1. Benedict, E. B.: *Endoscopy, as Related to Diseases of the Bronchus, Esophagus, Stomach, and Peritoneal Cavity*, Williams and Wilkins Company, Baltimore (May) 1951.

2. Jackson, C., and Jackson, C. L.: Bronchoesophagology, W. B. Saunders Company, 1950.
3. Terracol, J.: *Les Maladies de L'Oesophage*, Masson and Comany, Paris, 1951.
4. Cooper, W.: Pemphigus of the conjunctiva, *Ophth. Hosp. Rep.* 1:155, 1857.
5. a. Lever, W. F.: Pemphigus Conjunctivae With Scarring of the Skin. Report of Three Cases, *Arch. Dermat. & Syph.* 46:875, 1942. b. Lever, W. F.: Pemphigus Conjunctivae with Scarring of the Skin. Report of Three Additional Cases, *Arch. Dermat. & Syph.* 49:113, 1944.
6. Lever, W. F.: Pemphigus, A Histopathologic Study, *Arch. Dermat. & Syph.* 64:727, 1951.
7. Adam, C.: Untersuchungen zur Pathologie des Pemphigus conjunctivae, *Ztschr. f. Augenheilh.* 23:35, 1910.
8. Thost, A.: Der chronische Schleimhautpemphigus der oberen Luftwege, *Arch. f. Laryng. u. Rhin.* 25:459, 1911.
9. Thost, A.: Ueber Schleimhautpemphigus, *Arch. f. Laryng. u. Rhin.* 31:599, 1917-18.
10. Imperatori, C. J.: Stricture of the Esophagus Caused By Pemphigus, *ANNALS OF OTOLGY, RHINOLOGY AND LARYNGOLOGY* 40:1192-1193, 1931.
11. Baumann, F.: Essential Shrinkage of the Conjunctiva (Ocular Pemphigus) With Involvement of the Mucous Membranes of the Nose, Throat and Larynx and Stenosis of the Esophagus. (Detailed Treatment of a Chronic Disease of Unknown Etiology) *Med. Clin. N. A.* 17:795-804, 1933.
12. Greither, A.: Chronische Aphthose von Mundschleimhaut, Speiseröhre und Genitale mit stenosierender Atrophie und sekundärer Carcinombildung, *Hautarzt* 2:547, 1951.
13. This patient was presented before the New England Dermatological Society on February 9, 1949: Lever, W. F.: A Case For Diagnosis (Benign Pemphigus of the Mucous Membrane?), *Arch. Dermat. & Syph.* 61:1067, 1950.

DISCUSSIONS

DR. FRANCIS L. LEDERER (Chicago, Ill.): The literature contains a huge array of names concerning this symptom-complex.

Since we have no positive test for pemphigus, it is hardly likely that one could call these diseases pemphigus when, for example, there are ulcerative syndromes of the pharyngeal membranes under various names, like Bahcet's disease, Stevens-Johnson's disease, and Reiter's disease. Then we have that large group of collagen diseases identified with dermatomyositis and also scleroderma; to say nothing of some of the diseases that all of us have seen sometime in the Spring, such as erythema multiforme.

One of these cases described by Dr. Benedict fits in with Behcet's disease in that there were lesions in the mouth, on the conjunctiva, and in the vaginal vault. I can't begin to differentiate all of these because they react differently and some of them resemble one another.

What has not been mentioned is the resistance of these conditions to treatment—and even the use of ACTH and cortisone has had its advocates, but we

frequently have observed remissions after apparent cure. What is very important in the presentation, is the fact that in broncho-esophagology, there is a need to be also a dermatologist and to be aware of the diseases of the mucous membranes of the body.

DR. EDWARD B. BENEDICT (Boston, Mass.): I agree that these webs may not be specific. We can't prove that they are specific for benign mucous membrane pemphigus because we have webs in other diseases.

I fortified myself by having Dr. Walter Lever, who is a well-known dermatologist and who has written a book on the histopathology of many skin diseases, associated with me. He feels that the cases which we reported are definitely in the group of benign mucous membrane pemphigus, and that the webs seem to be like the adhesions seen in other parts of the body in that disease.

XCII

BLIND BOUGINAGE IN THE TREATMENT OF BENIGN ESOPHAGEAL OBSTRUCTION

LYMAN RICHARDS, M.D.

AND

HERBERT J. DIETRICH, JR., M.D. (By Invitation)

BROOKLINE, MASS.

At first thought the title of this paper might seem to constitute a flagrant disregard for one of the cardinal esophagological precepts of Chevalier Jackson, to whom I shall always be indebted for much of my fundamental training in endoscopy. I can see today as vividly as nearly 30 years ago the sinuous line of that brown pastel crayon as it traced a bougie's errant course toward a false passage of a stricture with perforation, and in those days almost certain death, as the inevitable outcome.

Thus I returned from Philadelphia to Boston with my esophagoscopic world before me, convinced that blind bouginage of any sort had no place in the treatment of strictures of the esophagus. A no less distinguished esophagoscopist than our President has condemned blind bouginage as "unwarranted and dangerous." When one considers what he, perhaps more than any other endoscopist, has contributed to our knowledge of the treatment of cardiospasm, having as far back as 1933 reported a series of over 800 cases,¹ one can scarcely dismiss such censure without a sound defense of blind bouginage and stout evidence in its support. I take, however, some comfort in the fact that it was only to blind bouginage as a diagnostic, not a therapeutic measure, that Clerf² had reference when he said "its hazards are so great as to make its use unwarranted."

It therefore came about during the first decade of my practice in which I treated a large number of children with various forms of esophageal stricture that I adhered strictly to this Jacksonian dictum and utilized nothing but retrograde bouginage, dilatation under esophagoscopic visualization, or olive tipped dilators passed on a guiding string. It was a profound shock when I discovered that certain older children, following gastrostomy for lye strictures, were returning to the surgical outpatient clinic for subsequent dilatation by

means of gum elastic bougies passed with only the sense of touch as security false passage or perforation, apparently without casualty.

It was during this same period that there was being carried out at the Massachusetts Eye and Ear Infirmary a far reaching clinical research into many of the problems relating to benign esophageal obstruction. Under the tireless energy and keen imagination of Dr. Harris P. Mosher³ a group of patients was subjected to painstaking investigation into the nature and cause of cardiospasm, esophageal webs and strictures of varied etiology, with a combined assortment of such procedures as bouginage, hydrostatic dilatation and direct esophagoscopy, all with supplementary roentgenological and fluoroscopic control. The ingenuity of the armamentarium and its clinical application to the many problems involved bear witness to Dr. Mosher's inventive mind and genius as an investigator, to which today we owe much of our knowledge of the pathology of benign obstruction of the esophagus.

This work had naturally led to the assemblage of a large number of patients whose lesions formed what has so often been somewhat crassly termed in medical literature, "clinical material." During the years 1925 to 1935 some 800 patients with benign esophageal obstruction were studied in this manner. These were not, however, all bona fide patients. Among them was an energetic and astute young hospital orderly who, for a financial consideration coming, I suspect, from Dr. Mosher's own pocket, allowed himself repeatedly to be bougied, fluoroscoped, dilated, barium mealed and otherwise esophagoscopically investigated as his personal gift on the altar of medical science. Years afterwards this business minded young man was wont to appear in the offices of various Boston laryngologists, requesting temporary and occasionally even permanent financial assistance, presumably still mindful of his past contributions to broncho-esophagology.

With the steady growth in the view that such esophageal lesions fall more properly within the scope of the thoracic surgeon than of the laryngologist, the number of these patients seen at the Infirmary declined. Many of those previously seen and treated had been essentially cured by conventional methods of dilatation, many had scattered to other localities and many had become lost to a follow-up in the manner of all such groups. There remained, however, a little band whose members had become what might perhaps be called treatment resistant and who returned periodically for dilatation as out-patients. They constituted what has come to be known as the Bouginage Clinic, for the conduct of which I found myself respon-

sible in 1941. These patients were all ambulatory, they had all had a preliminary roentgenological and esophagoscopy investigation and the nature and location of their lesion was known with reasonable accuracy. They returned to the hospital at longer or shorter intervals for dilatation of their spasm or stricture and many of them had for years been subjected to blind bouginage without the slightest suspicion of calamity. To have altered this well entrenched and apparently quite satisfactory routine would have meant either the tedious, and in my hands at least often unsuccessful, introduction of a string prior to dilatation or else repeated hospital admissions for esophagoscopies, entailing often impossible expense to the patient or an onerous load on already overburdened operating room facilities. Moreover, direct esophagoscopy has to me always entailed the handicap that even the largest esophagoscopes will often admit a bougie of quite insufficient caliber to meet the needs of the particular stricture. Thus a 10 millimeter esophagoscope, full lumen, will permit only a 20 #F bougie to pass through it.

For many of these patients their treatment necessitated departure from home at the crack of dawn, often a tedious train ride and an equally time consuming return, all to receive a treatment lasting only a few seconds. That many of them, despite proximity to fairly sized cities or towns, seemed unable to find any nearby facilities for such dilatation leads one to wonder why, with the steady expansion of the gospel of endoscopy and the constant training of new workers in this field, it was necessary for these patients to seek help at such expense of time and energy.

This practice has been continued under my supervision for 12 years with the full realization of the valid objections which may fairly be advanced against it. It has been adhered to first because, barring two incidents to which I shall refer later, there have been in some 5000 passages of bougies in this manner no accidents, to say nothing of fatalities and because the results, while not as spectacular as are often reported in the literature, have on the whole facilitated in these patients maintenance of a good nutritional state, a degree of comfort in the assimilation of food and an ability to swallow which have kept them satisfied and happy.

Admittedly such blind bouginage had best be eschewed by the heavy handed and the development of an intuitive sense of touch is a valuable asset. On more than one occasion attempts to pass a bougie beyond a point of undue resistance have been deliberately abandoned, only to find on a subsequent visit that the same or a larger bougie would pass with ease. It has been our impression, given

adequate preliminary study of the nature and location of a stricture, that it is an urge toward over-zealous dilatation rather than the mere passage of an unguided bougie which may lead to a catastrophe. Indeed we have felt that such guidance might even beget overconfidence and hence attempts to achieve a dilatation beyond the limits of safe dilatability.

Actually we have found only rare reports of esophageal perforation incident to blind bouginage, most fatal accidents recorded being associated with dilatations carried out under direct asophagosopic or hydrostatic dilatation under string guided control.

It was our constant experience throughout the course of these many bouginages to find patients eating and swallowing with perfect comfort in whom the stricture seemed resistant to all efforts at dilatation beyond a degree which at first thought might seem totally inadequate for comfortable deglutition. Thus, for example, one could often elicit no complaint from an adult whose stricture would not admit, with what seemed reasonable safety, a bougie of greater than a 26 or 30 F. caliber. It has been stated by eminent authorities in this field that any esophageal stricture is capable of dilatation up to the size of 45 F. at which point the patient may be considered cured and ready for dismissal. In the majority of our cases, exclusive of the patients with cardiospasm, we seemed to reach a maximum degree of dilatation beyond which almost endless repetition of bouginage was unable to effect any appreciable enlargement of the esophageal lumen and we were seldom able to achieve this idealistic figure of 45 F. It is hard to believe that New England strictures, despite this area's reputation for reserve and standoffishness, should be any more resistant than in the South and West. Perhaps we have leaned overly toward the contention that in the esophagus as elsewhere discretion is often the better part of valor and so have not pressed dilatation with sufficient vigor. We have, at least to date, never ruptured an esophagus. We continue, however, to be amazed that strictures of such small diameter appear so often to offer no impedence to the passage of a diet of normal food content provided that it is masticated with reasonable thoroughness.

It may well be asked why, if such a degree of dilatation achieves a satisfactory deglutitory status for these patients, they nevertheless return at intervals despite our failure to afford them any material increase in the size of the lumen of their stricture. The patients themselves provide the answer. Almost without exception when questioned as to the reason for their desire to continue treatment they have stated that they can maintain a comfortable status only

for a certain period of time, beyond which they experience an increasing need for dilatation. Since so often such dilatation, as noted above, adds little or nothing to an increase in the size of the esophageal lumen, one must conclude that even in long standing and presumably stable strictures there exists a tendency toward tissue contracture which will slowly increase if not counteracted by dilatation even if this does no more than maintain the status quo. Kindly attempts to dismiss such patients as needing no further treatment have been almost invariably met with the request that they be allowed to return when conscious of incipient dysphagia.

The contention that one encounters in the literature that such intermittent and nonprogressive dilatation is chiefly of psychic value is hard to accept when one considers the time loss and inconvenience involved not to mention the fact that such dilatation, however dexterously performed, can scarcely be regarded as a treat as well as a treatment.

The bougies used in the care of these patients have been of three types, the mercury-filled or Hurst bougie, employed almost exclusively in the dilatation of cardiospastic lesions in sizes from 42 to 60 F.; the classic gum elastic bougies for strictures in the cervical and thoracic esophagus; and the metal shafted or so-called Jackson bougie in occasional instances when treatment was carried out with the patient in the supine position.

Manifestly, none of these three types of bougie can be guided on a string. Neither the mercury bougie or any but the smallest elastic bougies can be passed through an esophagoscope, a situation equally true of the Jackson bougies except in the smallest calibers or in the very largest diameters of esophagoscope. Since both mercury and elastic bougies are advertised and manufactured by instrument makers of the highest reputation one must conclude that there still exists a demand for them and apparent sanction for their use by certain esophagologists who apparently do not consider their use unduly dangerous. They are the approved bougie now in use in the so-called Salzer method of preventing stricture of the esophagus following the ingestion of lye, but there is here of course no question of attempted dilatation, and thus of possible rupture, of cicatricial tissue. Oddly enough it was in such a case of swallowing lye with suicidal intent that there occurred one of the two known accidents incident to this period of 12 years of blind bouginage. An apparently uncomplicated passage of a mercury bougie in a woman of 35, who 10 days before had swallowed lye, was followed in six hours by obvious signs of an abdominal emergency. Immediate laparot-

omy revealed a perforation, not of the esophagus, but of the lesser curvature of the stomach. The tear was closed and the patient made a rapid and uneventful recovery.

In the other known complication, following passage of a bougie through a cervical stricture it was found impossible to withdraw it from the esophagus, as it were a, "bougie captivatus." No amount of traction was able to dislodge this accidental foreign body and since the patient manifestly could not be dismissed in this condition it was necessary to induce general anesthesia with intravenous sodium pentothal, following which the bougie was extracted without difficulty.

It is conceivable that over the years there may have been accidents of which we have had no knowledge but if so they have been unreported to the clinic to which such news usually gravitates with lightning-like rapidity.

A somewhat more detailed analysis of some 75 patients who at varied intervals have been returning for dilatation will, I believe, serve to illustrate some of the foregoing statements. They can best be divided into four groups: (1) Patients with a clear history of ingestion of some caustic substance with resultant cicatricial stenosis; (2) Patients with webs in the cervical segment of the esophagus; (3) Patients with fibrosis, sometimes of congenital but often of unknown etiology, and with obstruction occurring most often in the middle and lower third of the esophagus; and (4) Patients with terminal fibrosis, cardiospasm or so-called achalasia. The ages of these patients range all the way from eight to 80 years, but with an overwhelming proportion in the fourth and fifth decades of life.

CARDIOSPASM

It is manifestly quite beyond the scope of this paper to attempt any clarification of the long standing problem of the actual pathological etiology of this form of esophageal obstruction. Despite prolific clinical, anatomical and histological research, its true nature is still a source of debate and controversy. From a therapeutic point of view we have considered as cardiospastic patients those who showed the classical roentgenological picture of a dilated esophagus, ending in a narrow filamentous passage into the stomach and in whom large sized mercury bougies usually passed readily, often almost by sheer gravity, and without the feeling of resistance characteristic of fibrous strictures higher up in the esophagus. The dilatatory sensation is that of an obstruction which under gentle pressure relaxes and "lets go," permitting entrance through a lumen which approximates the normal esophageal diameter, rather than that of a tight constriction

which admits of dilatation grudgingly and with a tactile sensation of a lumen far short of that of a normal esophagus.

A survey of 20 patients carried on what may be termed the current roster and classified as cardiospastic shows them to have been under active if intermittent treatment for an average of eight years. During this period they have received an average of 60 dilatations per patient, the maximum number for a single patient in this time being 192. The mercury or Hurst type of bougie used in such treatments ranged in size from number 36 F to number 62 F, with an average diameter of 50 F.

On this admittedly prolonged system of treatment these patients have for the most part maintained a satisfactory body weight and nutritional status. Specific personal inquiry has again and again brought the response that without such intermittent dilatation they could not continue to eat comfortably and that they are convinced that only by such means could they maintain good health and enjoyment of life.

It should be pointed out that during the past 10 years other patients with cardiospasm, under a similar regime have been able to forego treatment and on this basis may be considered as cured. The failure of the 20 summarized above to reach a point at which they no longer need dilatation raises interesting questions. Perhaps in these patients initial hydrostatic dilatation was not pressed vigorously or persistently enough. Certainly reports such as those of Moersch and Vinson who in 70 per cent of the cases found hydrostatic dilatation completely effective lead me to feel somewhat remiss in this respect. Assuredly if such repetitious bouginage could be terminated by more drastic but effective methods both patient and doctor would be happier. Many of these patients had experienced years of deglutitory discomfort before we saw them and hence may fairly be regarded as having well established and chronic obstruction before the initiation of their treatment. With results which on the whole have been satisfactory it is easy to fall into a therapeutic rut and so to take a less zealous attitude toward more forceful methods of dilatation.

ESOPHAGEAL WEBS

Obstruction due to webs in the esophagus was most commonly noted in the cervical portion and was considered to be due in most instances to trauma of the mucosal surfaces with spurs of the cervical vertebrae sometimes playing an accessory role. Webs, more than any other lesion, were found amenable to direct esophagosopic treat-

ment by avulsion but unless such lesions are kept under observation and controlled by periodic dilatation there is a tendency toward recurrence of the same type of obstruction; hence the necessity for such patients to return to the clinic for treatment. Often the existence of such a web was first discovered through the accidental lodgement of some form of foreign body, the removal of which led to inquiry into the underlying pathology.

Sixteen patients in this series are classified as having had esophageal webs confirmed by previous esophagosopic and roentgenological examination. They too have been receiving dilatations for an average of eight years and the average number of dilatations per patient has been 63, figures curiously similar to those obtained in our analysis of the patients with cardiospasm. One patient with an esophageal web in nine years was given 137 dilatations. The average esophageal diameter maintained in this group in which elastic or more rarely mercury bougies were used was 37 F., a figure much smaller than in the cardiospastic cases.

It was strikingly evident that the patients under treatment for an obstructive web were, despite the number of their visits, generally freer of symptoms and more comfortable during their intertreatment intervals than those suffering from cardiospasm. Such figures would clearly seem to indicate that obstructive webs have an inherent tendency to recurrence unless periodically dilated at intervals of varying frequency.

LYE STRICTURES

The ingestion of lye or other caustic solution has long been a common but fortunately now less frequent cause of cicatricial stenosis of the esophagus. The majority of these lesions have, of course, been first noted in childhood but in our group there have been 12 patients averaging 37 years of age whose esophageal obstruction was found to have originated with the swallowing of some form of caustic solution. Several had had a previous gastrostomy followed by retrograde bouginage. The gastric stoma had then been allowed to close and subsequent dilatation has been carried on by peroral bouginage. In several instances the caustic had been swallowed many years prior to the first visit to the clinic during which time the patient had fought a constant battle with esophageal obstruction, meanwhile suffering varied interference with comfortable deglutition and making inconvenient concessions to a normal diet.

In this group there was no uniform site of predilection of the stricture which might be either cervical, mid-thoracic or suprahialal.

Multiple strictures were not uncommon and these presented by far the most difficult problem with respect to safe passage of a bougie without esophagoscopic or string guidance. Of these 12 patients each averaged four years clinic attendance and received an average of 30 bouginages per patient with attainment of an average lumen diameter of 34 F. If the time element of almost half that of patients with cardiospasm and webs is a fair criterion one may perhaps conclude that it is on the whole easier to dilate a lye stricture to the point of obviation of further treatment than in the case of obstruction due to webs or cardiospasm. It has been our impression that a mucosal cicatrix due to caustic ingestion is far less likely to involve the entire circumference of the esophagus, thus permitting dilatation of the remaining normal esophageal wall and hence achieving more rapid attainment of a lumen adequate for comfortable deglutition.

Such conclusions are admittedly subject to difficulties in closely following such patients who may unexpectedly return with further complaints from a lesion considered as essentially cured.

FIBROSIS OF THE ESOPHAGUS

The fourth and largest group in this series is that comprised of 25 patients with obstruction in the middle and lower third of the esophagus. The etiology of such strictures was often of obscure origin, sometimes obviously congenital, more often the sequel of some ulceration of the mucosa with resultant cicatricial stenosis of the lumen. Lues was noted as an occasional etiological factor, dilatation proving unusually stubborn and prolonged in such cases. Onset of symptoms was usually insidious and many of the patients had suffered for several years from varying degrees of dysphagia before seeking relief.

These 25 patients received dilatation for an average of ten years with an average of 43 dilatations per patient. One patient with a congenital stricture in 24 years was dilated 148 times, being still unable to tolerate more than a 26 F bougie. For the group the average maximum dilatation obtained during the period of treatment was a diameter of 36 F. Many of these patients returned to the clinic only at long and irregular intervals during which it was naturally presumed that they were symptom free. As with the other categories cited above such return was sometimes the result of accidental lodgment of a foreign body proximal to the stricture. Indeed certain of our patients first became aware of the existence of any partial esophageal obstruction because of such an accident and the removal of this transitory difficulty led to their disappearance from observation for indefinite periods.

It may well come as a surprise to many of you that such prolonged and repetitious dilatation of these various types of esophageal obstruction is either necessary or seemingly unavoidable. Perhaps we have lacked the courage to press dilatation sufficiently forcibly or frequently in order to attain a quicker and more permanent enlargement of the lumen to the point that such patients can be considered to need no further treatment. Against this justifiable criticism I have no strong defense. The patients with few exceptions have maintained a satisfactory nutritional level, and are almost unanimous in their opinion that without such periodic dilatation they could not eat comfortably and that they are greatly benefited by such treatment.

SURGERY IN BENIGN ESOPHAGEAL OBSTRUCTION

No discussion of esophageal stricture would be complete without at least a brief discussion of the possibilities of its surgical treatment. With the amazing achievements in thoracic during the past 15 years there have been opened up completely new concepts with regard to operative procedures on the esophagus heretofore regarded as fantastically difficult and hazardous. The idea of feasible anastomosis of the stomach to the pharynx once lay in the same dream world as jet propulsion and atomic fission. With increasing functional success and a steadily lowered mortality, it is not surprising that attempts to by-pass or even excise benign strictures are more and more finding a place in this field. It is obvious from the foregoing description of the tedious, time consuming and far from spectacular methods of dealing with such strictures by bouginage that any patient would welcome a more expeditious surgical procedure, given reasonable assurance of a safe and functionally satisfactory outcome. As far back as 1882 Mikulicz had opened the stomach and by manual dilatation of the cardia up to a diameter of four fingers sought the answer to cardiospasm. When one considers that a fully dilated hydrostatic bag is scarcely more than an inch in diameter it is not surprising that Mikulicz' almost obstetrical dilatation achieved the results he reported, as well as certain unhappy complications.

Oschner⁴ has stated that 25% of patients with cardiospasm do not respond to conservative therapy, to us a consoling observation, in view of the enviable success so often accredited to hydrostatic dilatation which we have seemed unable to duplicate.

Esophago-gastrostomy in the elimination of the obstruction incident to cardiospasm is now an established surgical procedure with results reported by Oschner as good in 90 percent of the patients but still with a mortality which time will undoubtedly lower. The

cardiospastic patient may perhaps still be in Hamlet's position of either bearing his present ills and electing bouginage or risking uncertainties of surgery despite its amazing and progressive achievements.

Still more hopeful is the prospect of complete excision of a stricture in the middle or upper third of the esophagus. Formerly successful only in dogs because of the problem of adequate blood supply, such removal of a short segment of the stenosed esophagus and end-to-end anastomosis has been successfully performed in a tiny infant by Gross.⁵ A major difficulty is the accurate preliminary estimation of the length of the stricture on which rests the feasibility of end-to-end approximation and which neither x-ray nor esophagoscopy will adequately provide. Esophago-gastrostomy has already been utilized in such high strictures but involves a more formidable procedure than in the case of cardiospasm. In advocating surgical excision of an esophageal cicatrix Gross stresses the often unyielding character of the scar tissue, a factor which has been so characteristic of many of our patients treated by bouginage. Several of our patients have been thus operated upon, too few from which to draw dependable conclusions. Undoubtedly the practicality of such operations will increase and more patients will be cared for in this manner.

Meanwhile the bouginage clinic goes on. Bit by bit its ranks, like that of the Grand Army of the Republic, are thinning out, some patients finding further treatment unnecessary, others gravitating to more distant areas, perhaps fortunate enough occasionally to fall into the hands of Dr. Moersch. There are fewer replacements, as the lure of thoracic surgical clinics becomes ever stronger. The remaining veterans, however, constitute a most interesting, grateful and appreciative group of patients.

REFERENCES

1. Moersch, H. J., and Camp, John D.: Diffuse Spasm of the Lower Part of the Esophagus, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 43:1165, 1934.
2. Clerf, L. H.: Dysphagia: Otolaryngological Aspects, *Trans. Amer. Academy of Ophthal. and Otolaryng.* (Nov.) 1949.
3. Mosher, H. P.: Notes on Esophageal Cases, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 43:1154, 1934.
4. Ochsner, A. J.: Surgical Disease of Esophagus, *Laryngoscope* 58:698 (July) 1948.
5. Gross, R. E.: Treatment of Short Stricture of Esophagus, *Surgery* 23:735, 1948.

DISCUSSIONS

DR. LOUIS H. CLERF (Philadelphia, Pa.): Mr. Chairman, I was anxious to hear Dr. Richard's paper for when I saw the words "blind bouginage," I immediately recalled my experiences in the bronchoscopic clinic under the tutelage of Dr. Jackson, who frequently called to our attention what Trousseau had said many years ago: "He who lives by the bougie will die by the bougie." He, of course, referred to blind bouginage.

I agree that esophagoscopy is blind bouginage, particularly when dealing with multiple strictures. Secondly, one is limited because a full lumen esophagoscope will admit a bougie (French scale) just three times the size of the lumen. In other words, a full lumen eight mm esophagoscope will permit the passage of a 24 bougie. When one wishes to pass a 40 bougie, one must see the plumber instead of manufacturers of surgical instruments.

I have practically given up esophagoscopy. Instead I have been utilizing olive-tipped fenestrated bougies that are passed over a previously swallowed thread.

I am not referring to the early case of lye burn but to the well-formed stricture. This is particularly necessary, as I see it, in the interest of safety if you have multiple strictures, for with a well swallowed thread, one can pass the bougie over all the strictures, whether there be one or a dozen. It's an out-patient procedure requiring, of course, attendance at the clinic. If one increases the size rapidly, probably one can dispense with this life time bouginage.

I believe firmly that blind bouginage still is a hazardous procedure because you have no notion where the bougie is going. There was a time when we gastrostomized all of our little patients in order to provide food, fluid, and also permit dilatation. Now we rarely do a gastrostomy because if the patient is still able to swallow mouth secretions, he can swallow a small black silk thread, and when that is gotten down, we tie a heavier thread to it and then giving the patient some barbiturate, we carry out bouginage with the patient dorsally reclined, using the olive tips.

In cases of achalasia I still use that old Plummer bag. I am interested to know how one can pass a mercury bougie in several of those patients that Dr. Richards had where the esophagus is S-shaped. I could see the opening of the hiatus by esophagoscopy and I wonder how the mercury filled bougie finds it.

I had a patient sent to me who had 82 dilatations with the mercury bougie, and his swallowing was just as poor after the 82nd dilatation as it was before he got his first. With the aid of a previously swallowed thread I used a Plummer dilator and after two dilatations he didn't find it necessary to come back for six months. So I felt that that was a decided advantage over any other method of dilatation that had been utilized in his case.

DR. MURDOCK EQUEN (Atlanta, Ga.): Dr. Richards is a man of great influence. A few years ago a paper of his was a great influence in discouraging young men from entering the specialty of otolaryngology. Again, his influence is so great that in many hundreds of cases the esophagus will be dilated by his method.

Dr. Clerf's reference to the Plummer bag reminds me that Dr. Plummer read an article at some medical meeting, at which one man got up and said that he had ruptured four esophagi with it and he didn't like it. Dr. Plummer replied that the reason he ruptured the esophagus was because he did not know how to use the instrument. That may happen to men who try this method of Dr. Richards.

However, in the case of the mercury dilator, one can watch it under the fluoroscope, and see it when it passes the cardia and enters the stomach.

Lye strictures, in children, are not so often seen in the south these days. Formerly, the principal use of lye in the home was in the making of soap, but now it is cheaper to buy the soap.

WALTER B. HOOVER, M.D. (Boston, Mass.): I am sure that careful observation will reveal many opportunities for the use of blind dilatation. Occasionally, the patient can be taught this method and can take care of himself, thus avoiding a trip to and from the clinic. This is especially true in the type of stricture in which the mercury dilator is effective because it is relatively safe to pass, not only for the doctor but for the patient as well.

Most dilatations with the large dilators through the esophagoscope are blind because the larger dilators obstruct vision. It would appear that almost everyone has given up the dilatation of an esophageal stricture to a size larger than the esophagoscope by the use of dilators passed through the esophagoscope.

I believe that there are indications for this greater dilatation and it can be readily accomplished by using a retrograde method through the esophagoscope. This is done by passing Jackson dilators, one at a time, through and past the stricture, then withdrawing them, one at a time, from below upward into the stricture and letting two, three or even four bougies rest in the stricture simultaneously. The smaller bougies may be drawn up into the stricture last. With various combinations of different sized bougies, quite a large dilatation can be made.

I have found this method of value in several cases in which there were firm, short strictures. I have not seen this method mentioned in the literature but believe it is worth mentioning in this discussion.

DR. HERMAN J. MOERSCH (Rochester, Minn.): After listening to Dr. Richards' most interesting presentation, I can arrive at only one conclusion: Dr. Richards must be a tremendously skillful surgeon.

Blind bouginage in the treatment of benign esophageal obstruction is associated with some degree of risk to the patient. Not infrequently patients come to Rochester who have mediastinitis and empyema that occurred after an attempted blind bouginage. This complication carries with it most distressing consequences. Although Dr. Richards and his associates were able to perform blind bouginage in such a large series of cases without a fatality, I doubt that the same results would be obtained in the hands of less skilled endoscopists. It appears to me that caution must be exercised in advocating a therapeutic procedure that, when employed by less skilled physicians, could lead to dire consequences.

The value of a thread in the guidance of a sound through an obstructed esophagus without danger of perforation can be best demonstrated by use of a thread tied to a post or other fixed object. The two fists are placed about the thread and a bougie with a wire spiral is passed over the thread through the two fists. The bougie will pass along the tortuous pathway without difficulty or the least trauma to the encircling fingers. When the same process is repeated without the thread as a guide, the difficulty of passage and danger of injury to the fingers and hands are immediately evident.

In most cases the patient can swallow a thread, provided it is twisted silk, without too much difficulty. The thread must be swallowed slowly or it will become tangled. Above all, the physician must have faith that the thread can be swallowed or the patient will not make a real effort to swallow it.

The procedure that Dr. Tucker described for the dilatation of tight strictures is excellent. Probably the greatest obstacle to the satisfactory dilatation of a stricture of the esophagus is failure to continue the procedure until the esophageal lumen is fully opened and adequately maintained. My associates and I strive to dilate the stricture sufficiently to allow passage of a 45 F. sound.

Use of the thread has been found to be of great value in dilatation for cardiospasm. Experience with approximately 4,000 cases of cardiospasm at the Mayo Clinic in the past forty years or so has demonstrated that about 70 per cent of patients who have cardiospasm can be completely relieved by adequate hydrostatic dilatation. In the remaining cases, the results are not satisfactory and in many of these it is necessary to resort to operation. The care of patients suffering from cardiospasm probably provides more gratification for the endoscopist than does care of any other group.

DR. LYMAN RICHARDS (Brookline, Mass.): I am well aware that the procedures which I have described are open to certain valid objections. It must be realized that the patients concerned represent a special group with long-standing disability and with a tolerance for the methods described which might not obtain in the average patient with esophageal obstruction. I would be the last to sanction methods which would be risky and I am quite in accord with the opinions expressed here by the discussors that by and large string guided or fluoroscopically controlled dilatation represents the safest procedure to be applied at the outset in undertaking the treatment of such patients. Our experience with the group here reported is therefore to be regarded more in the nature of the application of blind bouginage under very special circumstances than as an advocacy of its employment in place of more conservative and conventional methods in the average case.

XCIII

ESOPHAGITIS: A CLINICAL EVALUATION

WALTER B. HOOVER, M.D.

BOSTON, MASS.

The purpose of this paper is to record some of the more important impressions and experiences in the diagnosis and treatment of patients who have severe esophagitis of a nonspecific inflammatory nature, now often referred to as "peptic esophagitis," and to discuss, particularly, those patients who have become disabled by difficulty in swallowing, malnutrition, severe pain, hemorrhage and perforations.

A review of the case histories of 72 patients gives some insight into the clinical evaluation of these patients and emphasizes the fact that there is such marked variation in symptomatology, the pathologic process, accompanying disease and the nutritional state that each case must be individually studied and treated.

ETIOLOGY AND PATHOLOGY

The basic etiologic factor producing peptic esophagitis, peptic ulcer or ulceration is not understood. This unknown factor is often referred to as "the peptic ulcer diathesis."

Pathologic studies of biopsy specimens and the resected esophageal lesions do not reveal the etiologic factor which produces an esophageal ulcer or esophagitis. These studies and pathologic reports show nonspecific chronic inflammation of the esophagus, with ulceration and often granulation tissue. The mucosa of the esophageal wall is first affected and the inflammatory lesions spread to any depth through the esophageal wall and may involve its entire thickness. Occasionally, the esophageal wall may perforate or rupture spontaneously. Two separate ulcers with deep crater formation occurred in one patient. However, localized ulcers with *crater* formation are less common than diffuse inflammation and esophagitis. The lower esophagus is more frequently involved than other portions, yet the entire esophagus or any segment may be the site of esophagitis.

Inflammation or inflammatory reaction results in fibrosis and scar tissue formation with contracture and rigidity of the esophageal

From Department of Otolaryngology and Broncho-Esophagology, The Lahey Clinic, Boston, Massachusetts.

wall. Loss of function and stricture formation frequently follow these tissue changes.

The normal esophageal mucosa is protected from the active, acid gastric juices by constant tonic closure at the esophagogastric junction. Ulceration does occur in the esophageal mucosa and walls which, under normal circumstances, are not exposed to the active, acid gastric juices. Peptic esophageal ulceration appears to occur only when there is impairment of the sphincteric action which permits regurgitation of the gastric juices into the esophagus. Approximately 60 per cent of the patients who have disabling esophagitis have hernias through the diaphragm, most commonly at the esophageal hiatus and associated with a short esophagus. The herniation and the short esophagus decrease the effectiveness of the sphincteric action at the esophagogastric junction, but the short esophagus and diaphragmatic hernia alone are not sufficient to produce this condition. We have estimated that some 500 patients who have diaphragmatic hernias are seen in the Department of Gastroenterology in the course of a year and only a very small percentage of these have clinical esophagitis, much less the severe type which is now under consideration. In the routine gastrointestinal examinations, regurgitation is seen to take place frequently, without clinical or roentgenologic evidence of esophagitis. Whether or not a diaphragmatic hernia is present, regurgitation of acid secretions alone is not always sufficient to produce esophagitis. Treatment with ACTH and cortisone has been observed to have an untoward effect on the ulcer patient. Perforations of existing ulcers have occurred and new ulcers have developed.

Vomiting and increased intra-abdominal pressure may be factors in producing esophagitis. Three cases of esophagitis have appeared after, or have been associated with, pregnancy. In two of these cases, the stenosis which resulted from the esophagitis was severe enough to require dilations for a few months in order to relieve the obstruction. In the third case, considerable bleeding occurred as a result of the esophagitis but this case cleared up under medical management without dilation.

The indwelling Levin tubes used in the treatment of patients after abdominal operations appeared to be a related etiologic factor in four instances. In this respect, the elevation of the patient's chest and head and the drainage of the stomach by opening the Levin tube to prevent the acid secretions from regurgitating and remaining in the esophagus after abdominal operations must be considered to prevent pooling of gastric secretions in the esophagus.

Severe esophagitis is frequently associated with peptic ulcer, especially of the duodenum. Either an active ulcer is present or evidence of a previous ulcer is demonstrated by the roentgenologic finding of a duodenal cap deformity. In four cases of severe esophagitis, there was a history of a perforated peptic ulcer.

Of the many patients who have peptic ulcer and who are seen and treated in the clinic annually, only a small percentage have peptic esophagitis. Therefore, peptic esophagitis does not develop in many patients who have "the peptic ulcer diathesis."

Age has a definite bearing on this disease. Of the 72 patients, only two were under 30 years of age. The youngest was 20 years of age and had symptoms for five years before becoming disabled; the oldest was 80 years of age. Three-fourths of the patients were 50 years of age or older. Some of the older patients gave a history of symptoms dating back 30 and 40 years before they became incapacitated. It might be surmised that the patient's decreased vitality associated with age caused the tissues to become more vulnerable or that the aged are unable to bear the burden of this affliction. Certainly, debilitated states predispose to esophagitis.

Although esophagitis is more common in men, there is no great sex preponderance.

Surgical excision of the esophagogastric junction must now be considered an etiologic factor of great and increasing importance in the production of esophagitis. To date, a practical foolproof surgical method has not been devised which will establish a satisfactory opening from the esophagus to the anastomosed stomach or intestine and prevent regurgitation of secretions into the esophagus. Clinically, I have observed esophagitis after resection of the esophagogastric junction and esophagostomy for benign and malignant lesions when there was no evidence of "the ulcer diathesis." Esophagitis also may follow total gastrectomy with esophagojejunostomy as a result of the regurgitation of alkaline digestive fluids into the esophagus.

SYMPTOMATOLOGY

There is marked variation in the symptoms of patients who have severe esophagitis. Substernal distress, so-called "heartburn" or dyspepsia and various degrees of pain are nearly always present. In many, the distress is brought on or made much worse by lying down, stooping or bending. Many patients have found the pain so great on retiring that they have been obliged to seek relief by sitting in a chair in order to sleep.

Dysphagia has been an almost constant symptom in these severe cases, but may vary in degree, from slight difficulty with solid foods to complete obstruction to all foods and even liquids. Three patients had gastrostomies performed before they came to the clinic for treatment; others were maintained on intravenous feedings. The large percentage, however, were able to take some fluids. Occasionally, a patient came to the clinic because of an acute obstruction from a bolus of food or a foreign body caught in the constricted area.

The inflamed esophageal wall is often very irritable and muscular tone or muscle spasm may be increased markedly. It may be difficult to evaluate or distinguish the degree of obstruction caused by a spasm from the obstruction caused by inflammatory induration, both of which produce dysphagia and esophageal obstruction.

Spasm of the esophageal musculature may bring about intermittent obstruction and even obstruction of several days' duration. These spasms may produce or be accompanied by pain, which can be excruciating. Increased muscular tone or spasm of the longitudinal muscle may shorten the esophagus and be a factor in the formation of hiatus hernia.

Bleeding of a minor degree is common when regurgitation or vomiting takes place in patients in whom considerable obstruction and granulation tissue are present. This minor bleeding, although not severe, is alarming to the patient.

Continued bleeding, although not obvious to the patient or severe at any one time, may lead to severe anemia and consequent disability, as is illustrated by the following case. A man of 53 came to the clinic primarily because of excessive fatigue. Surprisingly enough, he had only minor epigastric distress without actual dysphagia. He had such marked dyspnea on exertion that he could not work. On examination by his physician, the patient was found to have a hemoglobin of 30 per cent, and the anemia did not respond to treatment. Roentgenographic studies at the clinic revealed a diaphragmatic hernia. The esophagoscopy examination demonstrated marked esophagitis, with bleeding throughout the middle and lower thirds of the esophagus. The ulceration was greatest in the mid-portion. The esophagogastric junction remained open and fluids regurgitated freely into the esophagus. In this case, the treatment consisted of a transfusion of 2000 cc. of blood and repair of the hernia. This patient has now been in excellent health for three years, and has a normal blood picture.

An alarming massive hemorrhage or repeated hemorrhage was a presenting symptom in eight cases. One recent dramatic case was referred to the clinic after thirty-two transfusions and balloon tamponade of the esophagus had been unsuccessful in controlling the bleeding. With further transfusion, a resection of the bleeding area of the esophagus and esophagogastrostomy were performed. The source of bleeding was an eroded artery in the mid-portion of the esophagus. While preparing for her hospital discharge on the fourteenth postoperative day, the patient died suddenly from an embolism.

In one patient who apparently had been satisfactorily managed by medical treatment and dilations, that is, she had been asymptomatic for six months, a massive hemorrhage occurred while the patient was touring and she died before effective therapy could be instituted.

Perforation or rupture of the esophagus associated with peptic ulcer occurred in three patients. The esophagus of one patient ruptured four months after diagnostic studies were done at the clinic; two were treated at the clinic; one died in the period before antibiotics were available and the other recovered after prompt antibiotic therapy, mediastinal and pleural drainage with repair of the perforation.

DIAGNOSIS

The diagnosis of esophagitis is not difficult if this condition is kept in mind and special esophageal examinations are made. A diagnosis could, no doubt, have been made months or years earlier in these cases if the physicians had insisted upon examination and if the patients had permitted examinations. These examinations consist of the fluoroscopic and roentgenologic studies which should be carried out by a competent roentgenologist. The presence of an interested esophagologist should make this examination more thorough and of greater value. The conclusion of the roentgenologist alone should not be accepted as final. An esophagoscopy must be done on patients who have symptoms, even though the roentgen studies are normal, if the early diagnosis of esophageal lesions is to be made. The esophagitis may extend over a considerable length of the esophagus. There is often a thin membrane with superficial ulceration and at the edge of the membrane there may be a red line or border. If the membrane over the superficial ulceration is removed, mild bleeding results. In cases of short duration, the induration of the esophageal wall may not prevent the passage of the esophagoscope. Much of the dysphagia

may be due to associated spasm. Good results may be achieved in a relatively short time under adequate treatment. In the cases of long duration, when there is dense cicatrix formation and marked induration, it is frequently impossible to pass the esophagoscope into the stricture and under these circumstances, granulation tissue, as well as quite active bleeding, may be present. The smaller bougies usually can be passed into the stricture but the lower portion of the esophagus cannot be visualized. In the cases in which gastrostomies have been done, retrograde esophagoscopy makes it possible to visualize the lower portion of the stricture.

In some cases, the esophageal walls are sufficiently indurated and thickened to prevent the esophageal lumen from closing. This permits fluids to pass and regurgitate as well, but prevents the passage of solid foods.

A great responsibility rests upon the esophagologist as these patients are in the cancer age group and must be suspected of having cancer. The esophagologist must do his utmost to rule out malignant disease. The experienced esophagologist can be relatively certain, in most cases, that the lesion is only an esophagitis. When the esophagoscope cannot pass the stricture and the area cannot be visualized, either within or beyond the stricture, a biopsy specimen may be obtained beyond the visualized area by the careful passage of forceps into the strictured region. Treatment and dilation often are necessary before the strictured area can be passed and visualized. The passage of a Levin tube through the strictured area may make it possible to place barium in the lower level and visualize, by means of roentgenograms, the area through which the barium solution would not pass in sufficient quantity. A tube of this type can be used for feeding purposes and treatment, that is, for medication and aspiration of acid secretions.

It must be remembered that a biopsy specimen in which cancer is not revealed does not rule out cancer. In one case, indurated ulcers that did not respond to treatment were resected. Since cancer does occur in some old cicatricial lesions of the esophagus, the cicatrices produced by esophagitis and esophageal ulcer should be looked upon with suspicion. The fact cannot be too strongly emphasized that many of these patients are treated over a period of many months, some even for years, and any new symptom or change again brings up the question, "Is cancer present?"

I also wish to re-emphasize that many of these patients have concomitant or related disease. A careful medical evaluation should

not be neglected. Restoration of fluid and electrolyte balance are often necessary and transfusions may restore the blood proteins and hemoglobin to the anemic, malnourished patients.

TREATMENT

Briefly, medical management consists of a diet which is adapted to the narrow constricted esophagus. It must be free of coarse or rough particles, finely divided, composed of soft, albuminous foods as used in the standard ulcer diet and given at frequent intervals.

Antacid therapy in the form of non-absorbable, non-irritating substances such as liquid aluminum hydroxide gel should be used frequently and may be used through the night also. Frequent feedings are important as they reduce the acidity or buffer the acid gastric juices.

Antispasmodics and drugs which decrease the vagus activity may be helpful. Roentgen therapy over the stomach has been used to reduce acidity when other methods have failed.

One important factor especially must be stressed and that is the prevention, in so far as possible, of the regurgitation of the gastric juices into the esophagus. Many patients who have esophagitis cannot lie in a horizontal position without suffering severe pain and these patients should always rest and sleep with the chest elevated. The patients are instructed in making or obtaining a back rest to be placed under the mattress so that the shoulders are elevated from 8 to 10 inches above the buttocks. This procedure has added great comfort and aided in healing these patients.

Weight reduction or other instructions which tend to reduce the intra-abdominal pressure should be applied whenever possible to prevent gastric juices from being forced into the esophagus.

Dilation to prevent stricture formation, which results from healing of the esophagitis, or to relieve stricture formation already present, is an important procedure in the treatment of esophagitis.

The type of dilator to be selected is determined by the particular problems to be met. In cases of recent onset, the soft mercury dilators may be entirely adequate. In cases in which gastrostomies have been performed, the Tucker retrograde method is of value. In the more severe type of stricture, I most frequently use the Plummer dilator over the swallowed thread which is used as a guide. This is a relatively safe, convenient method of dilation even in dense fibrous strictures and is much more effective than the mercury bougie for firm strictures.

Dilation with the Jackson type bougie, which is passed through the esophagoscope, can be carried out. However, it may be impossible to pass through the esophagoscope dilators which are sufficiently large to accomplish the desired dilation. Greater dilation with the use of Jackson bougies passed through the esophagoscope can be obtained by passing two, three and occasionally even four dilators past the strictured area one at a time; then pulling one at a time up into the strictured area from below. A number of observers have been impressed by the simplicity, effectiveness and better visualization and control obtained by this method of greater dilation through the esophagoscope.

Antibiotics are used in the treatment of severe esophagitis to reduce inflammatory reaction caused by secondary invading organisms and as a prophylaxis against the spread of infection that may occur as a result of manipulation.

THE SURGICAL TREATMENT OF ESOPHAGITIS

There is no unanimity of opinion concerning the proper surgical procedure and there is no standard procedure for all cases. Here, again, the operation should be carefully selected to meet the specific anatomical condition and be commensurate with the operative risk and the surgeon's ability.

There are two surgical emergencies: The first is esophageal perforation or rupture and the second is uncontrolled esophageal hemorrhage.

Except for the two emergencies, surgical treatment can be withheld for at least a time. Gastrostomy or jejunostomy for the purpose of feeding the patient should be extremely rare. Surgical exploration to make a diagnosis should likewise be rare, for the esophagologist should be able to dilate most strictures and make the diagnosis in most instances without resorting to surgical aid.

The repair of the diaphragmatic hernia in selected cases in which the esophagus is not greatly shortened and especially when the diaphragmatic hernia is of some size, may be markedly beneficial. Operations for the relief of the stricture and for decreasing the gastric acidity are varied and multiple procedures may be employed. Esophagogastrostomy after resection of the strictured area has been used more than other procedures, often with gratifying results. In three cases in which an esophagogastrostomy was done, esophagitis and stricture recurred. This procedure combined with vagus section and resection of the acid secreting portion of the stomach, as advocated by

Wangenstein, may give further assurance of success. Operations have not been entirely successful because they do not re-establish a sphincter at the lower end of the esophagus to prevent the regurgitation of gastric juices or other secretions. The etiologic factors still exist after the stricture has been removed. The so-called "ulcer diathesis" remains. In an occasional case duodenal obstruction from old ulceration may be present. Much thought must be given to the surgical approach to this problem.

Many of these patients are poor operative risks; many are aged and have a short life expectancy. In these cases, the decision for surgical treatment must be carefully weighed as there is a mortality percentage that cannot be overlooked, and it may be expedient to continue medical treatment and dilations even though the patients are not symptom free.

RESULTS OF TREATMENT

When a patient had not had symptoms or required dilations for a period of two years or more, the results of the combined methods of treatment in these severe cases were considered excellent. Twenty-eight patients have resumed a normal life with slight dietary and positional restrictions and some patients have even disregarded these.

Twenty-three patients have had to continue dilations at intervals but follow dietary and medical management. These patients, however, have not become invalids as a result of the esophageal condition.

Fourteen patients have remained invalids or semi-invalids mainly as a result of the esophagitis. A few have remained invalids as a result of other conditions such as senility, arthritis, hemiplegia and cardiac disease.

Seven deaths occurred, two as the result of perforation of the esophagus, two as a result of massive untreated hemorrhage and three were postoperative deaths.

SUMMARY

Peptic esophagitis is a relatively common condition. The severe disabling types are far less common. Many physicians and patients procrastinate until disabling symptoms force the patient to undergo diagnostic and therapeutic procedures which should have been carried out early in the course of the disease.

Most patients respond well to medical treatment with dilation. Most strictures can be controlled in a fairly satisfactory manner by appropriate dilation if the patients are willing to carry on with these

procedures. Long continued dilation may be necessary when surgical treatment is not accepted or is contraindicated. A few patients will rebel at the inconvenience and discomfort of continued dilations and desire surgical intervention.

Surgical treatment should be directed toward removal of the stricture and the acid secreting portion of the stomach or at least decreasing the acid secretions which contribute to the production of esophagitis. Medical management after operation should be continued, especially measures to prevent the *reflux* of acid gastric juice into the esophagus.

Impressions have been summarized from a review of 72 case histories of patients with disabling peptic esophagitis.

DISCUSSIONS

DR. WILLIAM A. LELL (Philadelphia, Pa.): Although Dr. Hoover has amply stressed the importance of the esophagoscopy examination in patients with benign inflammatory lesions of the esophagus, I would like to stress this point because many of these patients are in the age group in which malignancy is possible. Occasionally the diagnosis of carcinoma has been made on the basis of the x-ray findings alone and some of these patients have been referred to the surgical service for resection of the lesion without a preliminary esophagoscopy examination.

We all have had the experience that the original lesion showed the typical changes associated with a malignant process, but on repeated esophagoscopy examinations and biopsies this diagnosis could not be substantiated and the lesion was subsequently found to be inflammatory. Direct esophagoscopy inspection is an indispensable aid in differential diagnosis, irrespective of their radiographic appearance. Dr. Hoover has emphasized the fact that these lesions are not localized in one segment of the esophagus but may extend over a large portion, and may be located even in the upper esophagus, as shown by his illustrations.

Once the diagnosis of a benign lesion is made, the problem of treatment becomes important. Since the persistent inflammatory changes may produce fibrosis in the involved areas with subsequent stenosis of the involved segment of the lumen of the esophagus, dilatation either by direct esophagoscopy bouginage, mercury bougies or in-dwelling bougies, may be necessary to maintain patency of the lumen. Each individual case becomes a problem unto itself. By persistent effort these individuals can be aided to maintain adequate nutrition.

DR. HERBERT W. SCHMIDT (Rochester, Minn.): I should, again, like to emphasize two points. The first is the danger that exists when gastric juices are regurgitated into the esophagus. Dr. Wangenstein and others have shown that normal gastric juice placed in a dog's esophagus will produce ulceration. Clinically, it has been observed repeatedly that esophagitis which is severe enough to produce stricture of the esophagus develops in patients who have vomited over a prolonged period. Examples of this type of vomiting are the pernicious vomiting of pregnancy and the vomiting of patients who have obstructing lesions of the duodenum. In patients with a sliding type of diaphragmatic hernia, incompetency of the cardia is developed, and usually gastric acids are regurgitated into

the lower part of the esophagus. At the time of esophagoscopy, bile-stained fluid frequently is seen in the distal third part of the esophagus. This gastric juice has the potentiality of producing esophagitis and formation of ulcers. Ulcers at the esophagogastric junction may bleed or, if they heal, they may produce a cicatricial stricture which may interfere with swallowing.

The second point I should like to bring out is to urge thoracic surgeons to be conservative in operating on benign lesions at the cardia. We think that patients who have cardiospasm should be treated by means of dilatation, since in the great majority of cases excellent results are obtained thereby if the dilatation is done properly. Surgical intervention should be reserved for those patients who do not secure benefit from esophageal dilations. One of the complications seen after operations on the cardia for cardiospasm has been the development of an incompetent cardia with secondary esophagitis and formation of ulcers at the esophagogastric junction. When this occurs, a short esophagus usually develops. This complication is at times more distressing than the lesion that originally brought the patient to the physician.

DR. EDWARD B. BENEDICT (Boston, Mass.): I agree with the essayists about the importance of gastric juice, and believe that the explanation for the very high stenosis that Dr. Hoover mentioned is the presence of areas of gastric mucosa, even as high as those in some of the cases reported. I have biopsied similar very high lesions and found gastric mucosa in them.

WALTER B. HOOVER, M.D. (Boston, Mass.): I wish to thank the discussers. I do not have anything to add.

XCIV

CONGENITAL MALFORMATIONS OF THE TRACHEA,
BRONCHI AND LUNG

PAUL H. HOLINGER, M.D.*

KENNETH C. JOHNSTON, M.D.*

VICTOR N. PARCHET, M.D.†

AND

ARNOLD A. ZIMMERMANN, DR. ES SC.‡

CHICAGO, ILLINOIS

The relative frequency and sometimes grave significance of congenital anomalies of the respiratory system places them in a position of importance to the clinician, as well as the embryologist. While the anomalies may become apparent at any age, it is in the first weeks or months of life that they often present serious respiratory emergencies. Only a few papers survey this subject as a whole, although there are many reports concerning individual anomalies, such as congenital cysts of the lungs, and many single case reports of interesting, unusual congenital malformations.

This paper consists of a presentation of cases of congenital malformations of the trachea, bronchi and lungs, with particular emphasis on their clinical aspects.

CLASSIFICATION OF LOWER RESPIRATORY

TRACT MALFORMATIONS

The classification of the subject as a whole by Puzziel and Epifanio,¹ Gruenfeld,² and Jordan³ were considered. The latter was selected as the basis for this paper since it covered the field adequately; it has been simplified for practical purposes. Its original form includes all recognized anomalies as well as imaginable possibilities,

*From the Department of Otolaryngology, University of Illinois, College of Medicine; St. Luke's Hospital and the Department of Bronchoesophagology, Children's Memorial Hospital, Chicago.

†Otologische Universitätsklinik, Burgerspital, Basel, Switzerland.

‡From the Department of Anatomy, University of Illinois, College of Medicine, Chicago.

but omits anomalies such as monsters and conjoined twins. The adopted modified classification is as follows:

I. Anomalies of the trachea

- A. Agenesis or atresia
- B. Constriction or enlargement
- C. Tracheal evaginations or outgrowths
 - 1. Tracheoceles, diverticuli and cysts
 - 2. Fistulae
 - 3. Tracheal lung
- D. Abnormal bifurcation or deviation
- E. Other anomalies of gross morphology

II. Anomalies of the bronchi and lungs

- A. Complete or essential agenesis or atresia
- B. Constriction or enlargement
 - 1. Webs
 - 2. Compression from cardiovascular anomalies
- C. Bronchial evaginations or outgrowths
 - 1. Bronchoceles, diverticuli
 - 2. Congenital cysts
 - 3. Fistulae
- D. Subnumerary bronchi, lobes, fissures
- E. Supernumerary bronchi, lobes, fissures
- F. Anomalous bronchial and lung tissue attached to some part of the respiratory system
- G. Anomalous bronchial or lung tissue attached to tissues other than those of the respiratory system

REPORT OF CASES

This report is based on cases of various anomalies obtained from clinical and autopsy material of St. Luke's Hospital, The Children's Memorial Hospital and the Research and Educational Hospitals of the University of Illinois. Many of the cases had been hospitalized because of respiratory obstruction, often as acute emergencies, and therefore are of particular interest to the bronchogologist and are described from this point of view.

ANOMALIES OF THE TRACHEA

Agenesis and atresia of the trachea have been described,^{4, 5} but neither has been observed in this series. Since the condition is obviously incompatible with life, it would have little clinical significance.

Congenital constrictions or stenoses of the trachea have been reviewed in a separate communication.⁶ In all, 12 cases were presented and classified as suggested by Montandon.⁷ (a) Congenital fibrous strictures, consisting of webs, fibrous stenosis of tracheal segments, and tracheal stenoses associated with tracheo-esophageal fistula; and (b), stenosis due to absence or deformity of tracheal cartilages, such as tracheomalacia, individual cartilage deformity, and stenosis associated with anomalous vascular compression. Five cases were observed that were classified as fibrous strictures. One infant, 4 months of age, had a definite membranous web demonstrated on the lateral x-ray of the neck as being an hour-glass-shaped constriction of the trachea 3.5 cm below the level of the glottis. On inspecting this site with the bronchoscope, the small, central opening through the web was seen. Careful dilatation gradually increased the size of the lumen so that a tracheotomy was unnecessary. Another fibrous stricture of a segment of the trachea was seen in an eleven month old infant who was examined after tracheotomy elsewhere had been necessary to relieve continuous chronic respiratory obstruction. The obstruction was present in the upper part of the trachea above the tracheotomy tube and gradually responded to dilatation. The stenosis did not involve the larynx itself but began below the level of the cricoid cartilage. A third case of a congenital fibrous stricture of the trachea is included, although no endoscopic examination was made. The infant had an inspiratory and expiratory stridor and x-rays demonstrated a tracheal stenosis which persisted during both phases of respiration. No endoscopic procedure was made because of the fear of precipitating a tracheotomy, and, as the infant gradually improved, further work was deemed unnecessary.

Two cases of congenital esophageal atresia with tracheo-esophageal fistula are included in this series⁶ because of the finding of a fibrous stricture in the trachea. Both infants had a stormy post-operative course following the surgical anastomosis of the upper and lower segments during the first week of life. Their respiratory obstruction was a prominent feature of the post-operative period and continued to be a major problem during their first two years. Each had a stenosis at the point from which the lower esophageal segment had been removed from the trachea and the tracheal de-

hiscence closed. It is difficult to determine whether these are true congenital tracheal stenoses or whether they are the result of cicatricial contraction of the trachea at the point of the surgical closure.

Seven cases of congenital tracheal stenosis were considered in the group of stenoses due to absence or deformity of tracheal cartilages. Three infants demonstrated characteristics of tracheo-malacia. Symptoms of severe stridor with dyspnea and periods of cyanosis were manifestations of marked obstruction. In each instance the tracheal wall was exceptionally flaccid. The disappearance of symptoms with the passage of the bronchoscope through the narrowing, and the fact that symptoms disappeared with increasing age together with the bronchoscopic evidence of the flaccid tracheal wall indicated the presence of a localized retarded development of the tracheal wall. The flaccidity was more pronounced than that seen in the allergic infant and no evidence of allergy was present in these three infants. Lateral x-rays of the neck and chest demonstrated the stenosis visible on bronchoscopy.

Cartilaginous deformity as a cause of tracheal stenosis, exclusive of anomalies of the cricoid cartilage, was present in three cases. The stenoses in two cases were found at the level of the first tracheal cartilage. They were visible on x-ray and endoscopically were found to be cartilaginous in rigidity. The openings were posteriorly along the wall normally devoid of cartilage. The third case of this subgroup consisted of a funnel-shaped stenosis of the distal trachea with the point of greatest narrowing near the bifurcation of the trachea.

It is well recognized that congenital anomalies of the cardiovascular system may produce tracheal stenosis.^{8, 9} A previous report summarized the case histories of five infants and children in whom congenital anomalies of the aorta and its main branches produced severe tracheal stenosis as the predominating symptom.¹⁰ Eight additional infants have been seen in whom tracheal stenosis has been found to be caused by vascular constriction. The case histories of these additional cases are essentially the same as those presented in the previous publication. The symptoms are progressive dyspnea, generally reaching alarming proportions at four to five months of age, with dysphagia and cyanosis associated with feedings. In some, the dyspnea and dysphagia were present at birth. The significant finding in these infants has been the persistent deformity or absence of tracheal cartilages at the site of the vascular anomaly.¹⁰ Thus, the constriction is apparently not alone a compression stenosis, but is, in fact, an actual, persistent secondary deformity of the tracheal or bronchial cartilages, probably due to vascular compression in fetal life.

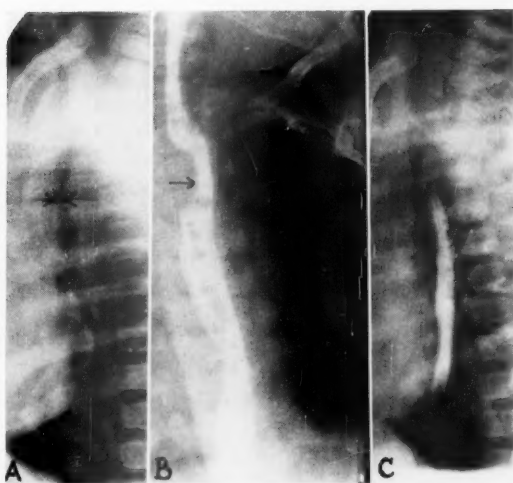


Fig. 1.—Tracheocele compressing the trachea and esophagus of a six months old infant. A and C, compression of the trachea. B, compression of the esophagus; the round shadow at the point of compression is the ossification center of the manubrium and does not represent the tracheocele.

Congenital enlargement of the trachea is rare as an isolated anomaly.¹¹ It has been observed in association with fibrocystic disease of the pancreas where the trachea as well as the bronchi are larger in diameter than normal, an observation recorded by Schneider¹² and confirmed in our examinations of these infants.¹³

Tracheal evaginations or outgrowths are subdivided into tracheoceles, fistulae and tracheal lungs. An example of the first category, a tracheal cyst, was observed in a six months' old infant who was admitted for study because of progressive respiratory obstruction, a wheeze, and a constant, brassy cough, together with considerable dysphagia. Fluoroscopy and films (Fig. 1) demonstrated a round mass posteriorly and to the left of the trachea obstructing the tracheal airway and compressing the esophageal lumen above the arch of the aorta. On bronchoscopy and esophagoscopy, the lesion was proven to be extraluminal. Exploratory thoracotomy (by Dr. Willis J. Potts) demonstrated the mass to be a cyst attached to the tracheal wall. Its removal resulted in a complete relief of symptoms. Histologic examination of its walls demonstrated respiratory epithelium. In two other cases, similar cysts were found attached to the bronchi and are described below. The tracheal cysts do not differ from the so-called bronchogenic or bronchial cysts.



Fig. 2.—Tracheal right upper lobe. The normal right upper lobe bronchus with its three branches is seen originating below the tracheal bifurcation. The bronchus of the tracheal lobe (arrow) originates on the right posterior-lateral wall of the trachea. There apparently is a large congenital cyst involving this lobe.

The most common of the fistulae that occur as anomalies of the trachea are the tracheoesophageal fistulae associated with congenital esophageal anomalies. They are seen most frequently in association with congenital esophageal atresia, although in an occasional fistula of this type, the esophageal lumen may be normal. A total of forty-four cases of tracheoesophageal fistulae were reported in detail elsewhere¹⁴ and sixteen additional cases have been seen since submitting the previous report; this indicates the relative frequency of this type of anomaly.

Tracheal lobe, a tracheal evagination, is an anomaly probably more common than one would suspect from a review of the literature. Huizinga and Smelt¹⁵ describe numerous variations of this anomaly, all associated with abnormal development of the right upper lobe. In its simplest form, the otherwise normal right upper lobe bronchus originates above the level of the bifurcation of the trachea and gives rise to three normal divisions. In another form, a complete duplication of this bronchus and lobe are seen, with a normal right upper lobe bronchus originating just beyond the bifurcation and an abnormal bronchus originating in the trachea. More commonly, however, the tracheal evagination consists of only the apical segment; in



Fig. 3.—Congenital abnormal bifurcation of the trachea with anomalous tracheal lungs. The patient has a dextrocardia; the right and left upper lobe bronchi apparently originate in the trachea. The bronchi descending from the tracheal bifurcation lead to a middle and lower lobe bronchus on each side.

some cases the right upper lobe has its normal three segments, in others the single apical segment originates in the trachea and the upper lobe bronchus gives rise to only the anterior and posterior bronchi and segments. An accessory right upper bronchus and lobe originating from the trachea has been observed in four cases in this series. There were no symptoms associated with the anomaly in three of the cases, and they were merely incidental observations on bronchograms. Two were adults, one male, one female, and two were infants with one of each sex again represented. One of the infants, 3 months of age, was admitted to the hospital acutely ill with what appeared to be pneumonia involving the right upper lobe. Acute symptoms subsided with antibiotic therapy but a large, round shadow persisted on x-ray until it gradually cleared following bronchoscopic aspiration, leaving a large cyst which appeared to involve the entire right upper lobe. A bronchogram (Fig. 2) demonstrated a normal right upper lobe bronchus with its three normal branches, and an accessory tracheal bronchus apparently leading to the congenital cyst.

Congenital abnormal bifurcation of the trachea, together with anomalous tracheal lungs presents the most interesting of the gross anomalies of the respiratory tract. Two cases are considered in this category. In one, previously described,⁶ the bronchus leading to

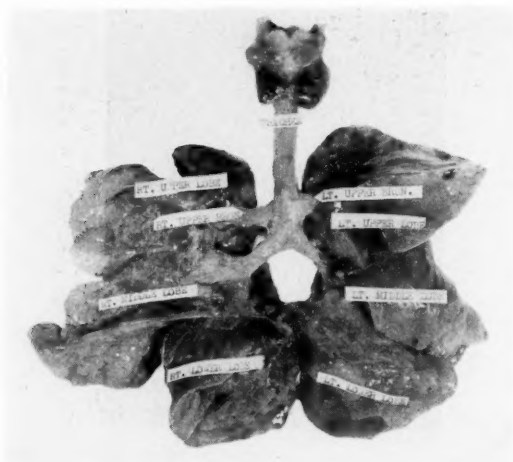


Fig. 4.—Post-mortem dissection of the bronchi and lungs of the same patient shown in Fig. 3.

the right lung was found to originate on the medial aspect of the left main bronchus, proximal to the level of the left upper lobe orifice, but distinctly distal to the normal position of the carina. This observation was made bronchoscopically and confirmed on exploratory thoracotomy, a procedure which had been performed to relieve dyspnea from tracheal compression. In a second patient, both the right and left upper lobe bronchi originated from the trachea. The trachea then continued downward to give rise to both a right and left main bronchus, from each of which originated a distinct middle and lower lobe and bronchus (Fig. 3 and 4). This female infant, two months of age when first seen, died at seven months of age as a result of multiple congenital deformities; these included dextrocardia, malrotation of the intestines, single accessory digits, deformities of the pelvis and right foot, and congenital absence of the spleen. The anomalies of interest in this report are the three lobes of both the right and left lungs. The right and left upper lobes arose directly from the trachea while the bronchi to the right middle and lower lobes as well as the left middle and lower lobes arose from a single trunk. The latter then divided into the branches for the respective lobes.

ANOMALIES OF THE BRONCHI AND LUNGS

Complete agenesis of an entire lung was observed in three of our patients, the right lung being absent in two female newborn

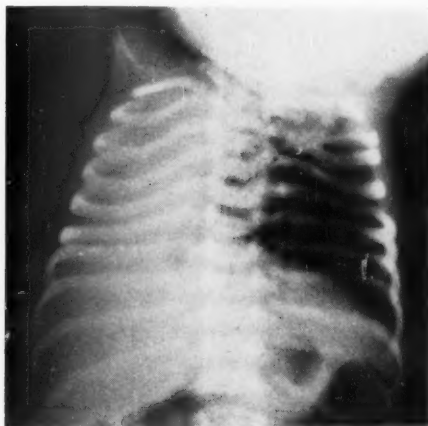


Fig. 5.—Apparent atelectasis of the right lung with shift of the heart and mediastinal structures to the right. Bronchoscopy for suspected obstruction of the right main bronchus demonstrated an absence of the right bronchus; post-mortem examination revealed a complete agenesis of the right lung and a rudimentary left lower lobe.

infants and the left in a seven year old boy. Both newborn infants had apparent atelectasis with dyspnea, shift of the heart into the right chest and density of the entire right thorax. These cases were reported with a complete review of the literature.^{16, 17} The findings were considered as indications for bronchoscopic study in the hope that secretions obstructing the right main bronchus could be aspirated to permit aeration of the right lung. On bronchoscopic examination a right bronchus could not be identified in either case and subsequent post-mortem examination showed an absence of the right lung and its bronchus. Numerous other congenital anomalies such as a horseshoe kidney, and double rib were present. In one of the two infants, the left lower lobe consisted of only a small tag of fibrous tissue without a bronchus.

The third patient, seen first in the Chevalier Jackson Clinic at Temple University and again at a later date at St. Luke's Hospital was originally suspected of having a foreign body obstruction of the left main bronchus. Demonstration by bronchography of a rudimentary left bronchus and the right bronchus rotated in a counter clockwise direction to partially fill the left hemithorax suggested the diagnosis of agenesis of the left lung.¹⁸ The absolute authenticity of this case as an agenesis of the lung might be questioned since the chest film and the bronchogram are identical to those previously

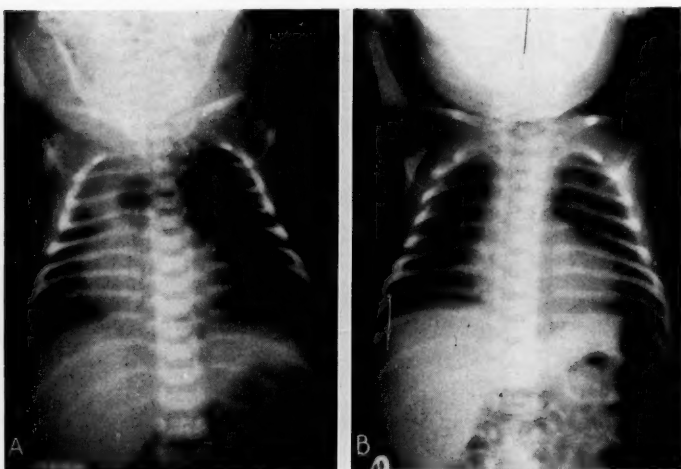


Fig. 6.—(a) Infant 24 hours old with obstructive emphysema of the left lung; a congenital bronchial web was found in the entrance of the left main bronchus. It was dilated with bronchoscopic forceps. (b) Chest film 24 hours later showing return of heart and mediastinum to the midline with equal aeration of both lungs.

reported cases of fracture of the bronchus.¹⁹ However, there is no history of chest trauma and re-examination of a chest film taken when the boy was one year of age showed the condition to be present at that time; these facts have led to the conclusion that this case is one of pulmonary agenesis. Angiograms or surgical exploration would be necessary to confirm the diagnosis. As there are now no clinical symptoms and general development has been normal, such studies have been deferred.

Constriction or enlargements of the bronchial tree follow the pattern of similar anomalies of the trachea.²⁰ Congenital atresia, as reported by Bonnier,²¹ was not observed in this series. Congenital webs appear as membranes partially or completely obstructing the bronchial lumen to produce obstructive emphysema or atelectasis.²² In one infant, 24 hours old, such a web was found at the entrance of the left main bronchus. It accounted for an extreme obstructive emphysema of the entire left lung (Fig. 6). Dilatation of the pin-point opening through the web resulted in immediate relief of symptoms. The over-expanded lung deflated and the heart and mediastinum returned to their normal positions.

Bronchial compression, either partial or complete is also produced by anomalies of the heart and great vessels.^{8, 9} Such constrictions have caused some of the extreme emergencies in this series. These lesions always enter into the differential diagnosis of respiratory obstruction in infants. Detailed descriptions of early cases of this type were presented in a previous report.¹⁰ Cardiac anomalies compressed the left main bronchus, while anomalies of great vessels were responsible for compression of either the left or right main bronchus. In one subsequent case, obstructive emphysema of the right lung was found to be caused by an enlarged heart which had also produced atelectasis of the left lower lobe. Other vascular anomalies were responsible for additional cases of left bronchus obstruction.

These conditions are of great clinical importance because they are responsible for some of the respiratory emergencies that occur in infants. They are of special interest in this review because examination of the bronchi demonstrates not only compression stenosis but also an absence or gross deformity of the bronchial cartilage in the segment of the bronchus involved. This deformity, which most likely occurred during embryonic development as the result of the shaping influence of the constricting vessel, persists to some degree after the vessel is severed. This is significant in that it explains a failure of complete relief of symptoms in some cases following surgery.

Two cases of bronchomalacia may be recorded in this group. These gave rise to rapidly progressive respiratory distress which necessitated an emergency thoracotomy and lobectomy in one instance; in the second case, the hypoplasia of the bronchial cartilages was an incidental post-mortem finding. The first patient, (C. M.) a two month old female infant had had episodes of wheezing, dyspnea and cyanosis during the previous month. On admission to the Children's Memorial Hospital, there was severe dyspnea and cyanosis with physical and x-ray evidence of extreme emphysema of the right middle lobe and atelectasis of the right upper and right lower lobes. On thoracotomy, an absence of part of the cartilaginous wall of the right middle lobe bronchus was demonstrated which was responsible for the emphysema through a check-valve mechanism. The essential findings were confirmed on subsequent pathologic study of the resected lobe. The infant made an uneventful recovery after lobectomy.

Bronchial evaginations or outgrowths consist primarily of fistulae, bronchogenic cysts, diverticuli and bronchoceles, which are relatively rare, and the more numerous congenital cystic malformations of the lungs. The bronchogenic cysts manifest themselves by varying de-

gress of localized bronchial obstruction. In one such case observed, the cyst obstructed the left main bronchus of an infant eight months of age.²³ A diagnosis of localized compression obstruction of the lower trachea and left main bronchus was based on x-ray examination, and confirmed by bronchoscopy. The cyst was then resected by Dr. Willis J. Potts. It measured approximately 5 cm by 2.5 cm and was situated in the mediastinum. It was attached to the left main bronchus just beyond the tracheal bifurcation and its removal left a 5 cm opening into the left bronchus which was closed by sutures. The infant made an uneventful recovery.

This type of cyst, together with the tracheal cysts described above is usually found to lie in the posterior mediastinum and is considered to be formed by the pinching off of a small bud or diverticulum of the foregut.^{23, 24} They contain a clear, thick, viscid fluid which produces pressure on both the airway and the esophagus (Fig. 1). However, since the walls are lined with ciliated pseudostratified columnar epithelium containing mucous glands and occasional plaques of cartilage, it is probable that they are the result of a pinched off pulmonary bud. The final diagnosis is dependent upon the resection of the cyst and the histologic study of its walls.

Congenital cystic malformations constitute a large variety of lesions which present many symptoms, gross and microscopic findings. A differential diagnosis between congenital and acquired cysts is of clinical as well as academic importance; the differentiation is not always a simple one. King,²⁵ Pugh,²⁶ Adams,²⁷ and Potts²⁸ recognized the complexity of this problem. McGrath²⁹ made a further differentiation by considering cysts lined with stratified columnar ciliated cells as arising from the bronchial tree, while those whose walls are composed of compressed alveolar cells as of alveolar origin. In the cases reviewed here, the differential diagnosis between congenital cystic disease and the other types of congenital anomalies often depended upon bronchoscopic examination, particularly in the new-born, since similar physical and x-ray findings were seen in infants with congenital bronchial stenosis, tension cyst, cardiac or vascular anomalies compressing the bronchus, spontaneous tension pneumothorax and even the acquired cysts due to staphylococcus pneumonia.²⁸

In five cases, lung cysts were recorded in routine autopsy material in infants. They had not been suspected and were not related to the primary cause of death. In one three month old infant an infected congenital cyst was the cause of death. In one infant a large congenital cyst was present in an anomalous right upper tracheal lobe, described in detail above.

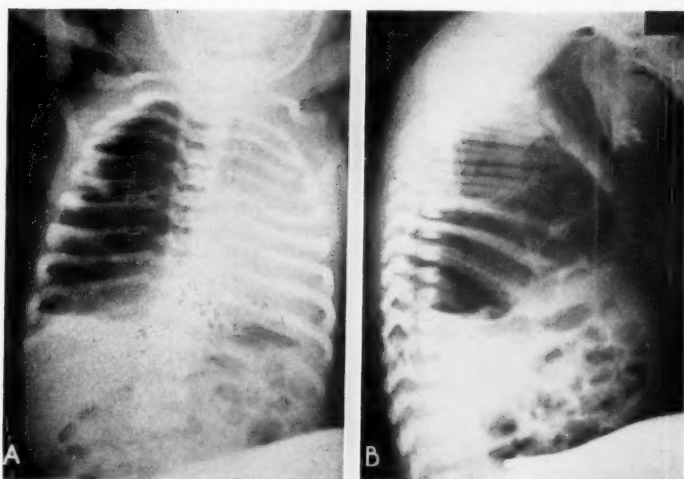


Fig. 7.—Congenital cystic disease of the right middle lobe of a five day old infant. The rapidly expanding cysts were responsible for acute dyspnea requiring immediate thoracotomy and lobectomy.

In three infants, one five, one eight and one fourteen days of age, large rapidly expanding cysts were responsible for acute, increasing dyspnea which required emergency management. Physical and x-ray findings demonstrated that a portion of the lung was rapidly becoming over-inflated, compressing other thoracic contents to the point of complete loss of function. The diagnosis in one of the three cases was obvious because of the appearance of the septa in the highly inflated right middle lobe (Fig. 7). This lobe had actually herniated into the left pleural cavity. An immediate thoracotomy and lobectomy were done with complete relief of symptoms. In the other two cases, bronchoscopic inspection preceded the thoracotomy and lobectomy because of the possibility of the presence of a congenital bronchial stenosis as the etiological factor of the obstruction.

The final differential diagnosis between congenital and acquired lung cysts cannot be made with absolute certainty without the histologic examination of the walls of the cyst itself. Lung cysts apparent during the first few days after birth probably are congenital. Cysts due to staphylococcal pneumonia, however, are truly acquired cysts. They develop in the immediate post-natal period and may simulate the conditions of congenital cysts. The majority of cysts are dilated branches of the bronchial tree. Histologically, the con-

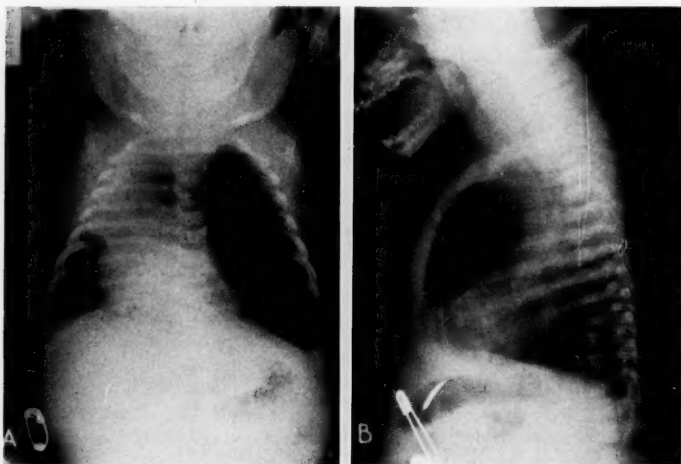


Fig. 8.—Idiopathic emphysema of the left upper lobe of an infant one month of age. No intrabronchial obstruction nor extrabronchial compression was demonstrated bronchoscopically. Severe respiratory distress was relieved by lobectomy of the emphysematous left upper lobe.

genital cyst is lined with ciliated columnar epithelium, while the inflammatory acquired cyst is lined with epithelium of a squamous character or merely with inflammatory exudates. The diagnosis is more difficult in older individuals when the congenital cyst may have become infected or may have been drained externally. This occurs frequently and may be erroneously diagnosed as an encapsulated empyema.

Of special interest are five cases of idiopathic emphysema of a pulmonary lobe. Such conditions may cause marked respiratory embarrassment. In two cases the left upper lobe (Fig. 8), in one the right middle lobe and in two cases the right upper lobe were involved. Lobectomy became necessary in these cases after bronchoscopy failed to demonstrate an intra-bronchial stenosis or extra bronchial compression. Symptoms consisted of progressive dyspnea and the findings were those of increasing lobar emphysema; these infants were all less than six months of age. Exploratory thoracotomy showed no vascular bronchial compression and gross and microscopic study of the resected lobes revealed no etiologic factors to explain the localized emphysema.

Cases of extreme emphysema of a lobe or of an entire lung, due to bronchial compression by an anomalous heart or great vessel, have been reported in detail elsewhere.¹⁰

Fistulae which result from bronchial evaginations are relatively common if one includes those cases of esophageal atresia in which the lower esophageal segment is connected with the left main bronchus rather than with the trachea. Differentiation of this type of secondary connection from that in which the connection was established with the lower portion of the posterior wall of the trachea has not been considered in this discussion.

One other case of a bronchial fistula is unusual and of considerable clinical significance. This new born infant was admitted to the hospital in extreme respiratory distress which was diagnosed as due to a tension pneumothorax. A tube was inserted into the pleural cavity at once. Symptoms were relieved but two days later the water seal was inadvertently broken and the infant expired in a few minutes. Post-mortem examination demonstrated that the right main bronchus did not branch normally, but communicated directly with the pleural cavity. Obviously, such a communication could and should have been closed surgically, but in view of the infant's critical condition on admission and its still precarious condition after insertion of the tube, further surgery was postponed. Post mortem examination then revealed the large congenital broncho-pleural fistula.

Subnumery bronchi, lobes and fissures are rarely reported in the literature.³⁰ While the most accurate method of determining the presence of these anomalies is the post mortem examination, the descriptions of the fissures, vessels and bronchi are usually inadequate or entirely lacking in most autopsy reports. Berg,³¹ Brock,³² Boyden,³³ Foster-Carter,³⁴ Scannel,³⁵ and Smith³⁶ describe subnumery variations of lobation and bronchi with lobar and bronchial variations seldom coinciding.

Autopsy reports of three premature infants in this series have shown varying degrees of pulmonary hypoplasia as subnumery lobes. In one, the left lung consisted of two rudimentary lobes weighing a total of 1.5 grams but a description of the bronchi was not given. In the second case, the left lung was hypoplastic, about 1/5th the size of the right, but the major bronchi were normal. In the third patient, an agenesis of the right middle lobe was apparent together with fetal atelectasis of the remainder of the lung, although again the bronchi were not described. In two additional male infants who died of other causes, one at six months and one at eight months of age, subnumery lobes were noted. In the former, the

right middle lobe was absent and, in addition, the infant had a hare lip and cleft palate. In the latter case, the upper and middle lobes of the right lung were lacking; in addition, a horse-shoe kidney and a patent foramen ovale were found. Again, unfortunately, the bronchi were not described.

Seven cases, two males and five females with subnumerary fissures are reported. They concerned the right lung in each case; the principle involvement consisting of only a great fissure on the right side in four cases and a complete absence of fissures in the other three cases. In most cases, this anomaly was associated with others. In one patient, a cleft palate, accessory spleen, dilated ductus arteriosus and patent foramen ovale were recorded; in another an accessory spleen was found; one had an extensive cardiac malformation and patent ductus arteriosus and another a tracheo-esophageal fistula. One infant had an imperforate anus, rectovaginal fistula, absence of gall bladder and horse-shoe kidney. The most interesting of the group, a premature, had no fissures on the right side, only two secondary bronchi to the right lung and an anomalous right pulmonary vein. There was a hypoplasia of the left lung, and, in addition, supernumerary thumbs.

Supernumerary lobes, bronchi and fissures. These may be grouped as tracheal accessory lobes, described above, and as lower accessory lobes. Mulse³⁷ classified these accessory lobe anomalies but included the azygous lobe, bronchogenic cysts and congenital cystic disease in this category, an arrangement not in agreement with embryologic concepts. An interesting case of a right tracheal lobe was that reported by Epstein,³⁸ in which an adenoma was found in the anomalous bronchus and bronchus and lobe were subsequently resected.

Lower accessory lungs were described by Davies³⁹ who concluded that supernumerary lungs or Rokitsky's lobes constitute one of the rarest anomalies in the chest, although about forty cases appear in the literature. It is suggested by Davies that the anomalous lung tissue in these cases is sequestered at about the seventeen paired somites stage, although it may occur much earlier. Grossman and Fishback⁴⁰ state that in about ten percent of normal individuals medial division of the left lower lobe takes place to form an accessory lobe. DeBaakey⁴¹ found the lower accessory lobe more common than the tracheal lobe and suggested that the anomalous accessory tissue was due to either a true third lung or to a sequestration of a part of the primitive pulmonary anlage. Bruwer, Clagett and McDonald,⁴² Steinert,⁴³ and Latienda⁴⁴ reported cases of an accessory lobe between the trachea and bronchi.

Supernumerary fissures without additional lobes are common surgical observations and are of little practical importance. Foster-Carter³⁴ found additional fissures to be the most frequent type of pulmonary abnormality, medial basal (infracardiac) and superior basal (dorsal) lobes being most frequently outlined as separate lobes.

Not included as accessory lobes, but considered rather as normal variability are the lateral divisions of the right upper lobe, present in about ten percent of all individuals, and the sup-superior (sup-apical) divisions of the lower lobes, present in about twenty-five percent of all individuals.^{45, 46}

Supernumerary bronchi, lobes and fissures are frequently observed during thoracic surgical procedures but not listed as such in diagnostic cross-filing. Eleven cases are presented in this report, eight males and three females. They are primarily autopsy findings of little importance clinically unless associated with bronchial anomalies. A distinct third lobe on the left side was present in each of these cases. In addition, an azygous lobe caused by the constriction of the right upper lobe by the azygous vein was present in two cases. In one case, a supernumerary fissure was present in the right lower lobe, although the bronchi were normal, and there were numerous anomalous vessels. One prematurely born infant had anomalous fissures in both lower lobes, separating the superior from the basal segments. An eight year old boy was found to have five lobes on the right side in addition to an azygous lobe, one anomalous fissure dividing the middle lobe and a second the superior segment of the lower lobe from the basal segments. The ages of these patients ranged from prematures to four adults, the oldest of which was sixty years of age.

The azygous lobe, of course, is not a true supernumerary structure. Therefore, it has not been considered as such in this review.

Thus, the most common of the subnumerary anomalies was a bilobation of the right side; that of the supernumerary anomalies, a trilobation of the left lung. The bronchial tree, however, was usually normal and these anomalies are of relatively little importance bronchoscopically, except for the interpretation of pathologic findings in the left middle lung field. They are of considerable importance for the thoracic surgeon, of course, in lobectomy and segmental resection.

Anomalous bronchial and lung tissue attached to tissues other than those of the respiratory system present a most interesting group of anomalies. The most striking example of this anomaly is the bronchus and lung originating from the esophagus.^{14, 47} This was observed in a five months old female infant referred for bronchoscopy because of what appeared to be multiple lung abscesses of the

left upper lobe. Bronchoscopy and bronchography demonstrated a fistulous tract leading from the mid-esophagus into the area of the multiple cavities. Subsequent surgical exploration and excision, as well as microscopic examination confirmed the suspicion that this lesion was an aberrant bronchus and lung originating in the esophagus.

Foster-Carter³⁴ described other anomalies belonging in this group. Disassociated tags of lung tissue appeared more frequently within the cavity than outside of it, often on the left side, associated with a defect of the left diaphragm. Jordan⁴⁸ added two similar cases of a cystic, infected, aberrant lobule removed by thoracotomy. Valle⁴⁹ described thirty-seven cases of aberrant lung tissue found in the abdominal cavity of which seven percent were associated with diaphragmatic hernia. Scheidegger⁵⁰ reported an anomalous lobe of lung, adherent to the left diaphragmatic pleura, which did not communicate with the respiratory system, but was connected to the stomach by a tract lined with gastric mucosa.

Atelectasis and atypical histology are conditions included in Jordan's classification of congenital anomalies of the human respiratory system. These will not be discussed here since this review is limited essentially to the gross anomalies. It is recognized, as Moersch⁵¹ indicated, that normally complete expansion of the lungs requires several post-natal days. This time factor is increased if the infant is premature. However, persistence of such an atelectasis may lead to bronchiectasis which would be indistinguishable from congenital bronchiectasis.

DISCUSSION AND SUMMARY

A series of 185 cases of congenital malformations of the trachea, bronchi and lungs is presented, collected from clinical and autopsy material of the Children's Memorial Hospital, St. Luke's Hospital and the Research and Educational Hospitals of the University of Illinois, College of Medicine, Chicago, from the year 1936 through 1951. The greatest majority of the cases, approximately 75%, (140) were seen at the Children's Memorial Hospital, where the total admissions during this 15 year period was 72,907. Thus, .002% of admissions to this hospital were found to have anomalies of the lower respiratory tract, exclusive of the larynx.

A classification of anomalies of the trachea, bronchi and lungs is suggested, based on the classification of Jordan; it has been simplified for practical purposes and serves satisfactorily from a clinical standpoint as a basis for study of this subject.

The most certain method of securing an exact diagnosis of congenital anomalies is through a study of autopsy material. However, in reviewing this material, it is noted that often descriptions are too short and frequently they do not include a correlation of the lobar, bronchial and vascular patterns. With the growing interest and improved technics of bronchoscopy and thoracic surgery in the new born, the need for a more accurate knowledge of the normal variations as well as the malformations becomes apparent in order to better assist in the interpretation of the clinical findings encountered.

The diagnosis of congenital malformations of the lower respiratory tract during life is made through the use of various services and the team work of the pediatrician, roentgenologist, bronchologist and thoracic surgeon. The history of obstetrical trauma or subsequent chest injury is important. From the physical findings one may differentiate a laryngeal anomaly from other obstructions to respiration by the character of the cry and the direct laryngoscopy. Physical examination of the chest is often the only method of interpretation of the x-ray film, when, for example, one must differentiate between an obstructive emphysema of one lung and an atelectasis of the opposite lung. A frequent erroneous interpretation of this type was noted when infants were admitted with a single chest film, the report stressing the atelectasis of the relatively uninvolved side rather than the much more significant obstructive emphysema of the opposite side (Fig. 8). Fluoroscopic study, inspiration and expiration films, the lateral view of the neck with the arms down and back to reveal the larynx, a barium swallow, bronchography, planography and angiocardiology all add significant information in special instances. Bronchoscopy without anesthesia is now an accepted diagnostic procedure in unusual respiratory conditions in infants. Limiting the duration of the procedure, the use of small instruments and the administration of oxygen through the scope during the examination all increase the margin of safety. Finally, exploratory thoracotomy in infants, now a feasible, well established procedure, increases the scope of diagnosis and adds therapeutic possibilities until recent years. It is because of this phase of the problem that the clinical, particularly bronchoscopic, study of the malformations here presented was undertaken.

As in all studies of congenital anomalies, the review of anomalies of the lower respiratory tract has shown a frequent association of anomalies of other organs and systems, as well as a frequent multiplicity of anomalies within the respiratory tract itself.

REFERENCES

1. Purriel, P., and Epifanio, C.: *Clasificación de las alteraciones Congenitas del Pulmon*, Hoja fisiol. 4:307-324 (Dec.) 1944.
2. Gruenfeld, G. E., and Gray, S. H.: *Malformations of the Lung*, Arch. Path. 31:392-407 (Mar.) 1941.
3. Jordan, H.: *Anomalies of the Human Respiratory System; A Proposed Classification*, Am. Rev. Tbc. 40:517, 1939.
4. Schneider, Louis: *Bronchopulmonary Hypogenesis*, Am. J. Med. Sc. 215: 665, 1948.
5. Marek, J. J.: *Congenital Deformity of Trachea*, Ohio State Med. J. 36: 1308 (Dec.) 1940.
6. Holinger, Paul H., Johnston, Kenneth C., and Basinger, Claire: *Benign Stenosis of the Trachea*, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 59:837 (Sept.) 1950.
7. Montandon, A.: *Congenital Stenosis of Trachea and Associated Malformations*, Pract. Oto-Rhino-Laryng. 6:179, 1944.
8. Newhauser, Edward B.: *Tracheo-Esophageal Constriction Produced by Right Aortic Arch and Left Ligamentum Arteriosum*, Am. J. of Roentgen. 62: 483, 1949.
9. Gross, Robt., and Neuhauser, Edward B.: *Compression of the Trachea or Esophagus by Vascular Anomalies*, Pediatrics 7:711, 1951.
10. Holinger, Paul H., Johnston, Kenneth C., and Zoss, Albert R.: *Tracheal and Bronchial Obstruction due to Congenital Cardio-Vascular Anomalies*, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:808 (Sept.) 1948.
11. Brenner, F., and Krauter, S.: *Ueber einen Fall von Besonderer Grosse der Luffrohre*, Wien. Klin. Wchnschr. 51:896-898 (Aug.) 1938.
12. Schneider, Louis: *Upper Lobe Bronchial Abnormalities Simulating Significant Pulmonary Tuberculosis (With Seven Illustrative Cases)* Radiology 55:390, 1950.
13. Holinger, Paul H., and Anison, George, *Fibrocystic Disease of the Pancreas: Bronchoscopic Observations*, II Congreso PanAmericano, Montevideo, Uruguay, 1950.
14. Holinger, Paul H., Johnston, Kenneth C., and Potts, Willis J., *Congenital Anomalies of the Esophagus*, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:707 (Sept.) 1951.
15. Huizinga, E., and Smelt, G. J.: *Bronchography*, Van Gorcum and Comp. Ltd., Assen, Netherlands, 1949.
16. Killingsworth, W. P., and Hibbs, W. G.: *Agenesis of the Lung; Review of the Literature and Report of a Case*, Am. J. of Dis. of Children 58:571, 1939.
17. Oyamada, A., Gasul, B. M., and Holinger, P. H.: *Agenesis of the Right Lung*. In press.
18. Jackson, C. L., and Bonnier, M.: *Technic of Bronchography*, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:771-785 (Sept.) 1937.
19. Holinger, P. H., Zoss, A., and Johnston, K. C.: *Rupture of Bronchus from External Trauma with Recovery*, Laryngoscope, 58:817 (Aug.) 1948.
20. Wolman, Irving: *Congenital Stenosis of the Trachea*, Am. J. Dis. Children 61:1263, 1946.
21. Bonnier, M.: *Un cas de stenose congenitale de la trachee avec bronche due lobe superieur droit atresiee et lobe superieur droit aplasique*. Imperformation de l'anus, Ann. med. chir. Hos. Sainte-Justine Montreal 5:13-18, 1947.
22. Wallace, James E.: *Two Cases of Congenital Web of a Bronchus*, Arch. of Path. 39:42, 1945.

23. Hardy, L. Martin: Bronchogenic Cysts of the Mediastinum, *Pediatrics* 108:113 (July) 1949.
24. Tucker, G.: Congenital Cyst of Mediastinum Producing Compression of Lower Trachea and Esophagus, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 55:693, 1946.
25. King, J. C., and Morris, L. C., Jr.: Congenital Lung Cyst, *J. A. M. A.* 274:280 (Jan.) 1932.
26. Pugh, David G.: Pulmonary Cysts, *Am. J. Med. Sc.* 208:673, 1944.
27. Adams, W. E.: Differential Diagnosis and Treatment of Congenital Cystic Malformations of the Lung, *Dis. of Chest.* 15:60, 1949.
28. Potts, Willis, J., and Riker, Wm. L.: Differentiation of Congenital Cysts of the Lung and those Following Staphylococic Pneumonia, *Arch. of Surg.* 61:684, 1950.
29. McGrath, Edward J., and Magnussen, M. J.: Cystic Disease of the Lung, *Arch. of Surgery* 57:427, 1948.
30. Anzola-Carillo, Intrapleurale, parapulmonale Bronchuscyste bei Fehlen des rechten Mittellappens, *Frankfurt, Ztschr. F. Path.* 54:118-125, 1939.
31. Berg, R. M., and Boyden, Edw. A.: An Analysis of Variations of the Segmental Bronchi of the Left Lower Lobe of Fifty Dissected and Ten Injected Lungs, *J. Thorac, Surg.* 18:216, 1949.
32. Brock, R. C.: *The Anatomy of the Bronchial Tree*, Oxford University Press, London, 1947.
33. Boyden, Edward A.: Cleft Left Upper Lobe and Split Anterior Bronchus, *Surgery* 26:167, 1949.
34. Foster-Carter, A. F.: Bronchopulmonary Abnormalities, *Brit. J. Tuberculosis* 40:111, 1946.
35. Scannell, J. G.: A Study of Variations of the Bronchopulmonary Segments of the Left Upper Lobe, *J. Thoracic Surg.* 16:530, 1947. Scannell, J. G., and Boyden, E. A.: A Study of the Variations of the Bronchopulmonary Segments of the Right Upper Lobe, *J. Thoracic Surg.* 17:232, 1948. Scannell, J. G.: Bronchographic Anatomy of Lungs, *Surg. Clinics of North America* 29:573, 1949.
36. Smith, F. R., and Boyden, E. A.: An Analysis of Variations of the Segmental Bronchi of the Right Lower Lobe of Fifty Injected Lungs, *J. Thoracic Surgery* 18:195, 1949.
37. Hulse, Wm. F., and Curtis, H. G.: Tracheal Accessory Lung, *American Review of Tuberculosis* 41:654, 1940.
38. Epstein, Isaac: Bronchial Adenoma in A Supernumerary Tracheal Lobe, *J. Thorac, Surgery* 21:362, 1951.
39. Davies, D. V., and Gunz, F. W.: Two Cases of Lower Accessory Lung, in the Human Subject, *J. Path. and Bact.* 56:417, 1944.
40. Grossman, J. W., and Fishback, C. F.: Pathological Condition in an Inferior Accessory Lobe and its Pleura, *Am. J. Roentg.* 63:43, 1950.
41. DeBakey, Michael, Arey, B., and Brunazzi, R.: Successful Removal of Lower Accessory Lung, *J. of Thorac. Surgery* 19:304, 1950.
42. Bruwer, A., Clafett, O. T., and McDonald, J. R.: Anomalous Arteries to Lung Associated with Congenital Pulmonary Abnormality, *J. Thoracic Surg.* 19: 957 (June) 1950.
43. Steinert, R.: Accessory Inferior Lobe and its Roentgenologic Significance, *Nord. med. (Norsk mag. f. laegevidensk.)* 5:95 (Jan.) 1940.
44. Latienda, R. J., Lobulo Pulmonar Accessorio Inter Traqueobronchial (A Predominio Bronquioloforme), *Rev. Asoc. Med., Ayent.* 60:954, 1946.
45. Jackson, C. L., and Huber, J. F.: Correlated Applied Anatomy of Bronchial Tree and Lungs with System of Nomenclature, *Dis. of Chest* 9:319 (July) 1943.

46. Parchet, Victor, Sprenger, F., and Mean, A.: *Über die Segmentanatomie der Lungen und ihre klinische Bedeutung.*, Archiv. Ohr-usw. Heilk. u. Z. Hals. usw. Heilk. Bd. 157, S. 365, 1951.
47. Gans, Stephen L., and Potts, Willis J.: Anomalous Lobe of Lung Arising from the Esophagus, Jour. Thor. Surg. 21:313-318 (Mar.) 1951.
48. Jordan, Hovey: Respiratory Malformations, Am. Rev. of Tuberculosis, Vol. 53, No. 1 (Jan.) 1946.
49. Valle, A. Roberto, and White, M. L.: Subdiaphragmatic Aberrant Pulmonary Tissue (Case Report), Dis. of Chest 13:63, 1947.
50. Scheidegger, S.: Lungenmissbildungen, Frankfurt Zsch. f. Path. 49:362, 1936.
51. Moersch, H. J.: Recent Advances in Bronchology and in Diagnosis and Treatment of Pulmonary Disease, ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY 58:1199-1210 (Dec.) 1949.

DISCUSSIONS

DR. W. LIKELY SIMPSON (Memphis, Tenn.): I think that as Dr. Holinger said, this and similar papers should be read before general meetings. It is important that the obstetrician, the pediatrician, the roentgenologist and the internist be well informed along this line.

DR. LOUIS H. CLERF (Philadelphia, Pa.): I am sure that Dr. Holinger has seen more cases of congenital malformation than I have.

I have seen some that should have been observed earlier. Obstetricians and pediatricians who see these infants when they exhibit their difficulties, should be fully aware that it is not merely a patient not wanting to breathe as he should, but that there may be a congenital anomaly present. The patient ought to be viewed from that standpoint and appropriate consultants called in rather than to have the pathologist find out what is wrong. That has happened too often.

OBSERVATIONS ON BRONCHIAL MOVEMENTS AND
ELASTICITY BY MEANS OF A RECORDING
BRONCHIAL CALIPER

ALBERT H. ANDREWS, JR., M.D.

CHICAGO, ILL.

Bronchial movements are frequently classified as angular deviation, lengthening and shortening, peristaltoid movements, and constriction and dilatation. The last type of movement is thought to be affected in some cases of bronchial asthma and not in others. Several small children have been bronchoscoped in which expiratory collapse of the trachea or main bronchus resulted in obstruction out of proportion to the clinical symptoms. Adults with marked pulmonary emphysema have been observed in whom the respiratory movements seemed very limited. These observations were interpreted to indicate a need for more critical observation of bronchial movements.

Studies of respiration as an indication of bronchial air flow have been made by different methods. Dennis Jackson¹ in 1917 reported a method in experimental animals of passing air through the trachea and recording changes in the volume of the lungs. Barach, Richardson, Cournand and their co-workers, Boothby and others, have presented many papers on bronchial obstruction by the functional respiratory examination which may be interpreted as an approach toward bronchial air flow characteristics. Tiffeneau and Drulel have interposed variable orifices in the air stream and developed data on the caliper of the total bronchial airway.²

Ellis,³ working in the Jackson Clinic, reported on tracheal and bronchial movements as recorded by balloons on the outside of the bronchoscopes.

These methods, while valuable, were thought not to give the precise information desired, so a bronchial caliper was devised.

From St. Luke's Hospital, Chicago, Illinois.

Supported in part by a grant from the Council on Physical Medicine and Rehabilitation, American Medical Association.

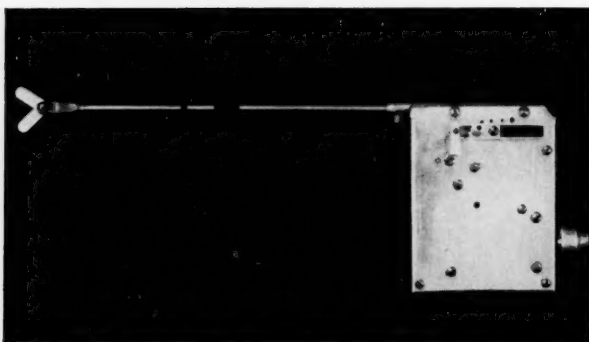


Fig. 1.—Photograph of the bronchial caliper. Part of the cannula which is 50 cm. long has been omitted. Blades are shown in the wide open position. The black dots are the four positions for the spring.

CRITERIA

The following criteria were developed for a bronchial caliper:

1. Atraumatic, expanding blades under variable spring control which would indicate the diameter of the trachea or bronchi and would follow respiratory or tussive movements.
2. Absence of airway obstruction and minimum interference with vision.
3. Size to permit passage through the seven mm standard Jackson bronchoscope.
4. Light in weight and freely movable within the bronchoscope to compensate for angular deviation and longitudinal motion of the bronchus in relation to the bronchoscope.
5. Construction to be such that mechanical failure of one part would not result in a potential foreign body.
6. No projecting parts which might interfere with rapid removal from the bronchoscope, regardless of the degree of spring tension.
7. Technique and speed of operation to be such that it could be used as a routine phase of the usual bronchoscopy.
8. Accuracy to be within plus or minus one millimeter and calibration of the instrument to be easily accomplished.
9. Capable of sterilization.

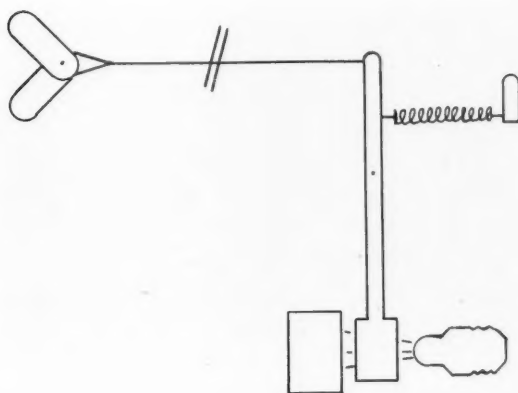


Fig. 2.—Schematic diagram of the mechanism of the bronchial caliper showing blades, linkage, spring, shutter, lamp and photo-electric cell. The beam of light and the photo-electric cell are actually at right angles to the shutter.

DESCRIPTION OF THE BRONCHIAL CALIPER

The first caliper incorporated an indicating dial, but the movements were too rapid to permit accurate reading. The second caliper activated a tambour which recorded on photographic paper by means of the Johnson Recording Oscillograph. The third instrument recorded electrically by means of an electrocardiograph machine. The final instrument records on a Brush Pen Writing Oscilloscope and seems to fulfill the above criteria.

The blades of the instrument consist of flat, polished, 1/32 inch thick, pivoted, rounded, aluminum strips, $\frac{1}{4} \times \frac{3}{4}$ inches (Fig. 1). The blades are opened by a linkage to the outside edge of each. The linkage consists of 0.010 inch iron wire of minimum elasticity which extends through a cannula into the case of the instrument, where it is attached to a lever. A small expanding coil spring is attached to the lever, and is adjustable to four different degrees of tension by an arm with a release lever (Fig. 2).

The lever is extended so as to interrupt partially a light beam focused on a photo-electric cell;* the light beam being produced by a lens end, miniature screw base, pocket flash light lamp. This system does not impose any friction or significantly additional inertia.

*Electrical design produced by the Universal Electronics Corporation, Chicago.

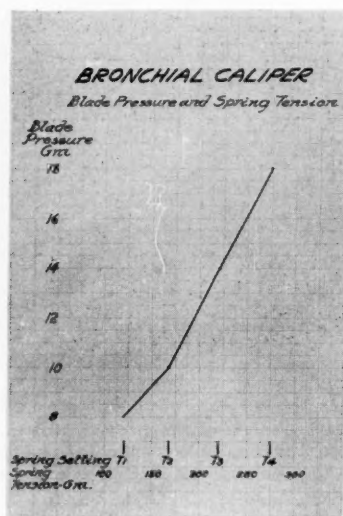


Fig. 3.—Chart of the spring settings and spring tension and the pressure exerted by the blades.

The spring exerts an opening force on the blades of 0, 8, 10, 14 and 18 grams plus or minus one gram depending upon the setting. The force is approximately uniform through the range of the blades except at the widest open position where the force is reduced (Fig. 3).

For calibration, the blades are drawn through a step gauge from 7.5 to 30 mm in diameter with steps of 2.5 mm (Fig. 4 and 5). Calibration curves (Fig. 6) show the relation between the actual distance across the blades and the ECG record at the different spring settings. It is noted that the relationship has good linearity, but that there is some variation between the different spring settings.

Pneumographic recording of respiration is made by means of an extendable, air-tight chamber applied to the lower chest and recorded by a tambour. With the electrocardiograph apparatus, the writing arm is placed so as to cast a shadow on the photographic paper. The Brush oscillograph is of the two channel type with the second channel used for the pneumograph recording. While this method for recording respiration has well known inaccuracies, it is adequate for identifying bronchial movements in relation to quiet breathing, deep breathing and cough.

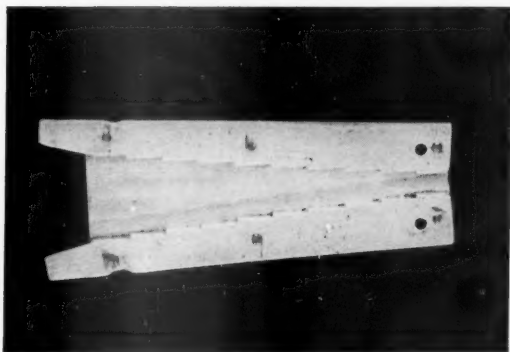


Fig. 4.—Photograph of the step gauge used for calibration of the caliper. The gauge is drawn over the blades from the small to the large end.

The instrument is dried following sterilization and oiled by means of a large diameter suction tube which is passed over the blades and sealed to the cannula by a split rubber stopper.

This instrument fulfills the criteria mentioned above. An improved model is under construction which incorporates the following modifications: wider blades for giving a wider area of contact and reducing indentation of the walls; detachable cannula for more convenient sterilization and substitution of different size and shaped blades; forcep handle with ratchet for ease of manipulation and control of spring tension, and wider variation in spring tension.

ABBREVIATIONS

Abbreviations used in this paper are as follows:

T1, T2, T3, T4: setting of the spring adjustment.

T2-1, T3-1, T4-1: difference in pressure exerted by the blades between T1 & T2, T1 & T3, etc.

DT1, DT2, DT3, DT4: diameter of the bronchus as determined at spring settings of T1, T2, etc.

DT2-1, DT3-1, etc: change in diameter of the bronchus as a result of the dilating force T2-1, T3-1, etc.

ET2-1, ET3-1, etc: numerical expression of the elasticity of the bronchus based on DT2-1, DT3-1, etc.

R: pneumographic record of respiration.

B.C.: bronchial caliper record.

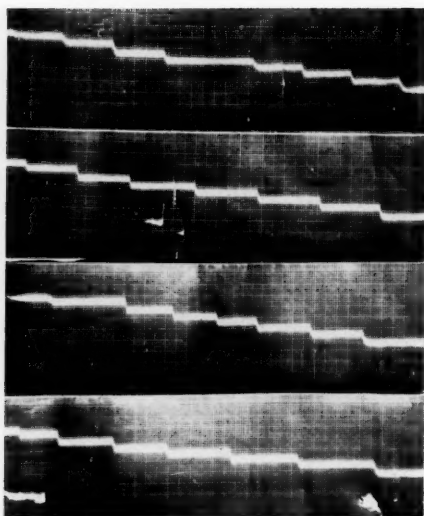


Fig. 5.—Calibration record for Case No. 3. Record in this and following figures read from right to left and from closed to open position of the blades. The distance between the thin vertical lines represent 0.04 seconds and between the thick vertical lines represent 0.2 seconds.

TECHNIQUE

With the pneumograph applied to the patient, and after the usual techniques in the bronchoscopy are completed, the bronchoscope is accurately lined up with the trachea or right or left main bronchus, and the head firmly held in this position. With the instrument attached to the recording apparatus, it is inserted into the bronchoscope and a short record is made to establish the base line. The recorder is activated by a foot switch under control of the bronchologist. The instrument is passed to a mark on the cannula which places the blades just beyond the tip of the bronchoscope. The spring tension is increased and a record made at each tension. The instrument is lightly supported so that it is free to move with the bronchus or trachea. An identifying mark is made on the record for each phase of the calibration. The patient may be instructed to take a deep breath and exhale forcibly or to cough. As the instrument is withdrawn, a check base line may be established. The procedure may be repeated for each area as desired.

The accurate lining up of the bronchoscope with the bronchus and angular deviation with respiratory movements are the major

BRONCHIAL CALIPER

Tension Calibration

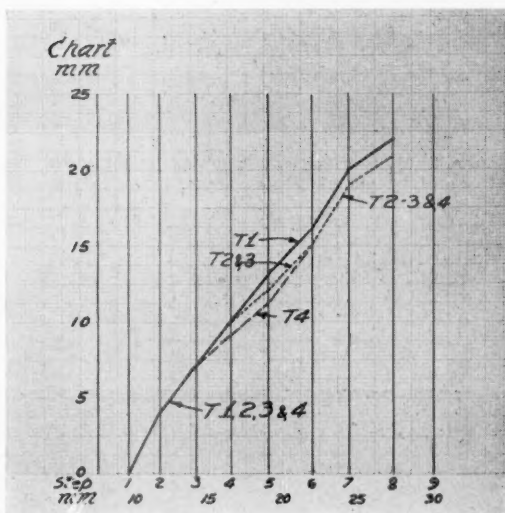


Fig. 6.—Chart of the calibration of the caliper. The heavy vertical lines indicate the actual diameter of each step of the gauge. Chart mm is the measurement from the electrocardiograph recording.

problems in this technique. They require meticulous attention and undoubtedly are the causes for most of the unsatisfactory recordings. The advantage of the pen writer oscilloscope is that it permits observation of the record as it is made in contra-distinction to the photographic type of recording.

Calibration curves (Fig. 5) are made before and after use. A graph is prepared similar to that in Fig. 6 and used for determining the actual bronchial diameters.

RESULTS

Table 1 records the results of bronchial calibration in nine cases and esophageal calibration in one patient, and Table 2 records the clinical data and functional respiratory examination. Records of bronchial movements associated with respiratory movements, deep breathing and cough are greater at the higher tensions and the reported observations are made at T4, unless stated otherwise. The

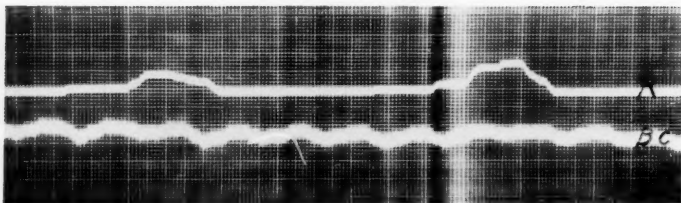


Fig. 7, Case 2.—Record from the esophagus at the aortic narrowing at T4, cardiac rate of 75 pulsations per minute.

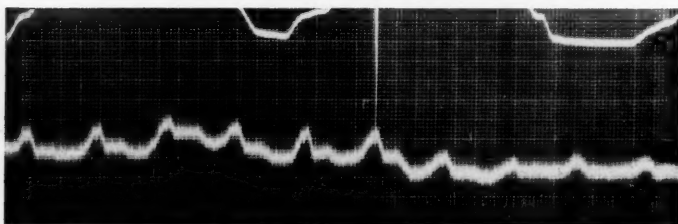


Fig. 8, Case 3.—Left main bronchus. Cardiac pulsations cause a change of diameter of 2 to 3 mm. Cardiac rate of 80. Stretching of the bronchial wall is shown by the difference in level between T3 and T4 of about 2 mm.

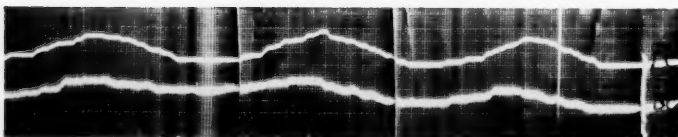


Fig. 9, Case 4.—Right main bronchus. Respiratory bronchial movements of 7 to 10 mm at T4 are shown.

TABLE 1.—BRONCHIAL CALIBRATION. MEASUREMENTS IN MILLIMETER. ELASTICITY. SEE DISCUSSION. RESP. INDICATES BRONCHIAL MOVEMENTS WITH RESPIRATION.

Patient Number	1	2	3	4	5	6	7	8	9	10
Rt. Main										
Esophagus	7	7	7	8		6.5		8		
DT 1	7	7	7	9		7		11		
DT 2	7	8	8	9		8		12		
DT 3	16	10	10	11		8		10		
DT 4	Puls.	7.9	7-12	7-9†		7.9	7-11†	11-12	7	
Resp.	7-10			6-12						
Cough				8-11				9-11		
ET 1-2	0	7-12	0	6-3		3.8		18.8		
ET 1-3	2.4	2.4	2.4	2.0		3.6		2.1		
ET 1-4	4.7	4	4.8	4.2		2.5				
Lt. Main										
Bronchus				7						
DT 1				9						
DT 2		7								
DT 3		7.5		10				7		
DT 4				8-11†						
Resp.		ET 3-4		14.3						
ET 1-2		2.4		4.8						
ET 1-3										
ET 1-4										
Trachea										
DT 1		22				20		26		2.7
DT 2						22		27		26
DT 3						24		28		28
DT 4		23				24		29		26-28†
Resp.		17-22			12-25	22-25			22	
Cough						5.0			12-22†	
ET 1-2						3.3		1.9		Unsat.
ET 1-3						3.3		1.3		0.6
ET 1-4						2.2		0.9		0.4
ET 1.		0.005								

†Bronchial Asynergy.

*Caliper placed at level of aortic narrowing.

††3.

TABLE 2.—CLINICAL DATA AND FUNCTIONAL RESPIRATORY EXAMINATION.

Patient	1	2	3	4	5	6	7	8	9	10
Age	56	23	35	46	14	43	47	61	44	61
Sex	M	M	M	F	M	F	F	M	F	M
Body Type	slim	Med.	Slim	Obese	Slim	Obese	Med.	Med.	Med.	Med.
Ventilation cc/min				6,740	9,120	6,230	7,780	11,400	8,290	11,860
Efficiency %				3.75	2.1	3.2	2.9	2.8	3.0	2.8
Vital capacity cc				2,880	1,950	2,300	1,285	2,095	1,245	2,175
Expiratory curve				Normal	Normal	+	+	+++	+	+++
Maximum breathing				49	39	47	14	36	23	19
Capacity L/min.										
Diagnosis	Crico. pharyn. dysphagia after laryn- gectomy	Tuber. abcess R.U. lobe	Suppur. bronch- itis	Catarr. bronch- itis	Bronch. asthma	Emphy- sema	Bronch. asthma	Emphy- sema, Heart disease	Emphy- sema	Emphy- sema

diameters are less than might be expected from the ease with which the 7 mm bronchoscope is inserted into the main bronchi. Elasticity is indicated by the increase in diameter which occurs as the blade tension is increased. The method of determining the numerical value of elasticity is given in the discussion.

The caliper is capable of recording transmitted impulses from the aorta in either the esophagus (Fig. 7), or the left main bronchus (Fig. 8). Observations on the shape of the pulsatory curve are not made.

Dilatation of the bronchi or trachea during inspiration and constriction during expiration or cough are assumed to be normal movements (Fig. 10 and 11). The opposite movements are thought to be indicative of bronchial asynergism, and were observed in patients No. 5 and 8, Fig. 11.

Normal movements of the trachea during continuous coughing are observed in Fig. 10, but the tracheal constriction did not follow all of the cough movements. This would suggest that the tracheal movements are slower than the respiratory muscle action, and that the recorded tracheal movements are not the result of intrathoracic pressure changes.

Increase in bronchial diameter as caused by increased pressure on the caliper blades is thought to represent bronchial elasticity, Fig. 8.

DISCUSSION

This work is in the nature of a preliminary report. A critical attitude has been assumed toward the records and as a result the number of patients selected for reporting has been limited. Patterns of bronchial size, movements or elasticity are not apparent from the work so far. The data reported has been made by electrocardiographic recording. A serious disadvantage to this method is the inability to see the record as it is being made. Drift of the galvanometer is present, although this is not disadvantageous in cardiac mensuration, it is a serious disadvantage in this application. The Brush pen writing oscillogram eliminates these disadvantages.

The speed of response of the caliper is pertinent to a study of bronchial movements. An estimation of this has been made by drawing the step gauge over the instrument at varying speeds. Using the oscillogram at high chart speed (125 mm per second), and the caliper at low and high tension, records were made (Fig. 13). At

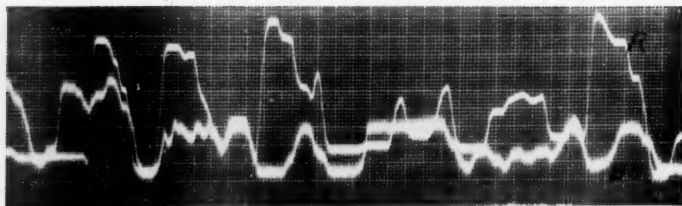


Fig. 10, Case 6.—Trachea during continuous coughing. Diameter 17 to 25 mm at T4.

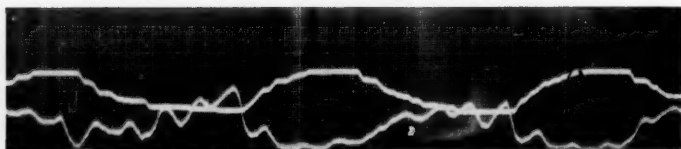


Fig. 11, Case 8.—Right main bronchus. Diameter 7 to 11 mm at T4. Bronchial constriction during inspiration and dilatation during expiration. This is thought to be bronchial asynergism in a patient with bronchial asthma as compared to normal movements as illustrated in Fig. 9.

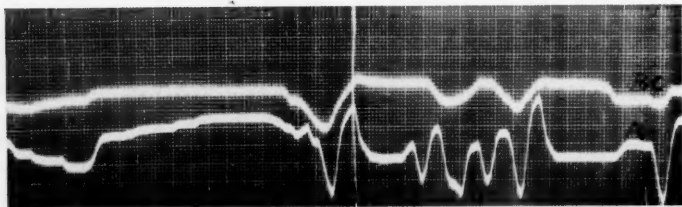


Fig. 12, Case 10.—Trachea. Diameter 12 to 22 mm during cough. The bronchial movements are not completely in phase with all of the cough movement.

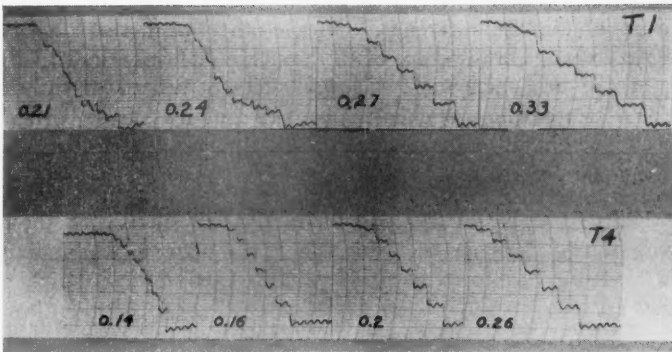


Fig. 13.—Tests on response speed at T1 and T4. The figures indicate the time required to draw the step gauge over the blades. Brush Pen Writing Oscillometer at a chart speed of 125 mm per second. The fine waves are 60 cycle induced current due to inadequate shielding. At T1, the caliper appears accurate at 0.27 seconds, and at T4, it appears at 0.16 seconds but not at faster speeds.

the lowest tension, the recordings were accurate when the gauge was moved over the blades in 0.27 seconds, but not in 0.24 seconds. At the highest tension, it was accurate in 0.16 seconds, but not 0.14 seconds. This speed of response is thought to be adequate for recording bronchial movements, although the speed of bronchial movements is not known.

It must be recognized that observations on bronchial movements at the level of the main bronchi may not be applicable to the smaller bronchi, and this may limit their value in diseases such as bronchial asthma and obstructive pulmonary emphysema. This concept may hold also for responses to broncho-dilator drugs which is a further application of bronchial calibration technique. The use of this technique in the smaller bronchi of the lower lobes and in children requires further mechanical refinements in the instrument.

Elasticity is defined as that property by virtue of which a body resists and recovers from deformation produced by force. Determination of elasticity of the bronchi interposes several problems to which a satisfactory solution has not yet been proved. Variation in the size of different bronchi cause variation in response to the stretching force even though their elasticity is the same. An expression of elasticity should compensate for this variation.

Force applied to the walls of the bronchi by the blades of the caliper may cause an elongation of the bronchial walls in the segment

being tested. The elasticity is related to the length of the bronchial segment, or the circumference. On the other hand, the force may result in a change in the shape of the bronchial lumen without elongation of the bronchial wall. This elasticity is related to the cross sectional area of the bronchial lumen. A combination of both may occur. The circumference is used in this report although it may not be the most satisfactory.

Elasticity is expressed generally by Young's modulus in the following formula⁴ where an elongation s is produced by the weight of a mass m , and force of gravity g , in a wire of length l , and radius r :

$$M = \frac{m g l}{\pi r^2} s$$

The force in this work is the difference between tensions at the various spring settings. The length of the elastic body is the circumference at the initial tension, e.g., $DT1 \times \pi$. The cross sectional area of the bronchial wall is beyond reasonable measurement or estimation, and is arbitrarily eliminated. The elongation of the bronchial wall in response to the force at the different tensions is expressed by $DT2-1 \times \pi$. Substituting in the above formula gives the following:

$$M = \frac{(T2-1) \times (DT1 \times \pi)}{(DT2-1) \times \pi}$$

Eliminating factors gives the following:

$$M = \frac{(T2-1) \times (DT1)}{DT2-1}$$

In this study, for the expression of elasticity, it is desirable to show a larger value as the elasticity increases, and so the formula is inverted. It is also desirable to have the expression close to unity so the factor of 100 is introduced. The formulas used are as follows, but it must be recognized that their adequacy is questioned:

$$ET2-1 = \frac{DT2-1}{(T2-1) \times (DT1)} \times 100$$

$$ET3-1 = \frac{DT2-1}{(T3-1) \times (DT1)} \times 100, \text{ etc.}$$

SUMMARY

1. Need for a means of measuring and recording the diameter and movements of the bronchi is indicated.
2. Criteria for development of an instrument for this purpose are presented.
3. A bronchial caliper is described which has expanding blades under variable spring tension and which records by means of an electrocardiograph machine or pen writing oscilloscope.
4. Data collected from 11 patients is presented and correlated with the clinical data and functional respiratory examination, and illustrative records are included.
5. An attempt is made to express elasticity of the bronchial wall by application of the physical principles of elasticity.
6. Because of the preliminary nature of this work, no conclusions are made at this time.

Acknowledgement and appreciation is expressed for the considerable aid given by Carl A. Johnson, M.D., on the application of the Johnson oscilloscope and the electrocardiograph apparatus.

REFERENCES

1. Jackson, Dennis E.: *Experimental Physiology and Materia Medica*, C. V. Mosby, St. Louis, Mo., 1917.
2. Tiffeneau, R. G., and Drulel, P.: *Méthode Bronchométrique pour l'étude du Calibre bronchique*, *Préss Méd.* 58:1186-1189 (Oct. 25) 1950.
3. Ellis, M.: *Mechanism of Rhythmic Changes in Caliber of Bronchi During Respiration*, *J. Physiol.* 87:298-309 (Aug. 19) 1936.
4. *Handbook of Chemistry and Physics*, 31st edition, Chemical Rubber Publishing Co., Cleveland, Ohio, 1949.

DISCUSSIONS

DR. CHARLES M. MORRIS (Philadelphia, Pa.): Of particular interest in Dr. Andrews' presentation were the tracings in cases of bronchial asthma. One undetermined factor, and one which presumably has considerable influence on bronchial calibre is that of intrathoracic pressure. Granted that there are variations in elasticity and rigidity of the bronchial wall, perhaps equally important, or even more important, are the alterations which occur in intrathoracic pressure.

It is commonly observed bronchoscopically that in pulmonary emphysema or in typical cases of bronchial asthma, there is marked expiratory collapse of the trachea and larger bronchi, and this presumably results from an increased expiratory intrathoracic pressure in the presence of diffuse peripheral obstruction. It is quite conceivable that deficient support in the walls of the smaller peripheral

bronchi or bronchioles might be a precipitating or contributing factor in this mechanism, along with the other recognized mechanisms of bronchospasm and obstructive secretions.

If such is the case, then it will be extremely interesting if data can be obtained regarding intrathoracic pressures at which the smaller bronchi collapse, or correlating changes in intrathoracic pressure with changes in bronchial caliber. This would appear to be a difficult problem, but one which might be productive of useful information.

This project has been a very worth-while one. There is probably no field of medicine in which understanding of physiology is more important in interpreting the manifestations of disease than in the respiratory tract. One has only to recall the early observations of Chevalier Jackson and others on the mechanisms of bronchial obstruction to realize that such work as that of Dr. Andrews may prove of great value.

DR. LOUIS H. CLERF (Philadelphia, Pa.): I would like to congratulate Dr. Andrews on his ingenuity; also, on his desire to further our knowledge of the normal and pathological physiology of the tracheobronchial tree.

Many have marvelled at bronchial movement. If Dr. Andrews can help us unravel some of our difficulties by getting some information concerning bronchial movements, also whether they really are movements of a passive character or whether there is some bronchial activity *per se* it would be of value.

If he can develop an apparatus that will give information concerning the variations of bronchial movements, the pharmacologists, will be delighted to work in this field. Many of their interpretations are on an empiric or clinical basis, neither of which are very scientific.

This is a fine piece of work and I hope he can explain some of his investigations and bring us at a later date some very positive evidence.

DR. PAUL G. BUNKER (Aberdeen, S. D.): Certainly there are many cases in which we would like to have accurate information such as Dr. Andrews is attempting to get from his studies of bronchial rigidity.

I have such a case, and Dr. Holinger described three or four similar cases in his discussion of unexplained emphysemas, in which it would be interesting actually to measure the rigidity of a bronchus where malacia is suspected. I believe that Dr. Andrews stated that he placed his expanding tips just below the end of the bronchoscope, and it seems to me that the rigidity of the bronchial wall would be influenced by the presence of the bronchoscope in such close proximity. Perhaps if the tip of the bronchoscope is maintained at a higher level and the tip of his gauge then placed accurately under fluoroscopic guidance a truer reading would result.

DR. ALBERT H. ANDREWS, JR. (Chicago, Ill.): I wish to thank the discussors for their kind comments.

The relationship of intrapleural pressure to asthma and emphysema and bronchial movements is an extremely stimulating subject. A technic, simpler than inserting a needle into the pleural space, is needed for measuring intrapleural pressures and I am sure it would give us very significant information, as Dr. Norris has implied.

Testing with drugs has not been done, but it should be pointed out that the bronchial calibration has only been done in the main bronchi. The instrument under construction at the present time will probably go into the lower lobe bronchi, but whether or not observations on bronchoconstrictor and particularly bronchodilator drugs will be significant at that level of the tracheobronchial tree is open to question.

Dr. Bunker's comments are well taken. The blades are usually passed so that they will just clear the bronchoscope. This places the blades between a half and three-quarters of an inch ahead of the tip of the bronchoscope. It may be that the bronchoscope does limit the bronchial movements. It would be nice to do it under fluoroscopic control, and I hope that we will be able to do so in the future. However, it makes a complicated procedure out of a technic that we have tried to keep at a clinical level.

HIDDEN OR UNSUSPECTED BRONCHIECTASIS
IN THE ASTHMATIC

RICHARD H. OVERHOLT, M.D.*

AND

JAMES H. WALKER, M.D.*

BOSTON, MASS.

Bronchiectasis, hidden and unsuspected, is often present in the asthmatic patient. Attacks of severe bronchospasm may be produced and maintained by the mere presence of bronchial deformity acting as an exciting cause. Bronchospasm can be more than a symptom of bronchiectasis—by obstruction it can produce bronchiectasis. Since it can be either cause or effect, consequently, bronchiectasis should always be suspected and looked for in bronchial asthma.

The asthmatic patients seen at the Overholt Thoracic Clinic have been classified only as medical failures. This group complains of severe attacks of shortness of breath, cough, and wheeze. Many of them have a history of purulent sputum, hemoptysis and repeated respiratory infection. Their previous medical therapy had included adrenalin, aminophyllin, expectorants, antibiotics, ACTH, cortisone, diet manipulation and change of climate. In addition, many had sinus surgery, or removal of nasal polyps, hemorrhoids, prostatic tissue, gall bladder or teeth. Such foci of infection are at times found to be the trigger mechanism causing bronchospasm, but eradication had not produced relief in these patients. Since cough, shortness of breath, wheeze, and production of purulent and bloody sputum are pulmonary symptoms, we believe that the search for a focus of infection should begin with a complete examination of the pulmonary fields.

Bronchial obstruction is chiefly responsible for the clinical manifestations of bronchial asthma. Diminution of the bronchial lumen by partially or completely blocked bronchi leads to impairment of ventilation and gaseous interchange which is followed by interference

*From the Department of Surgery, Tufts College Medical School.

of the circulatory dynamics. Obstruction may be due to bronchospasm, bronchostenosis, foreign bodies, or nodes either compressing or perforating the bronchus. These factors may operate alone or in combination to produce bronchiectasis. The bronchial obstruction is usually segmental and may be partial or complete, with emphysematous changes or atelectasis. The increase in the negative intrapleural pressure with its dilating effect, the inability to adequately raise bronchial secretions with resultant bronchial infection promotes the formation of bronchiectatic segments or subsegments. The infection or inflammatory changes may also produce definite fibrobronchostenosis causing bronchiectasis to develop in the distal portion of the segment. Prickman and Moersch¹ noted the presence of bronchostenosis bronchoscopically in 60 of 140 asthmatic patients. Hyperplastic and fibrotic lymph nodes resulting from repeated respiratory infection or pulmonary tuberculosis may encircle or compress the bronchi and produce bronchial obstruction. Brock² and Graham³ have pointed out that the anatomic architecture at the middle lobe take-off is most conducive to compression and obstruction by enlarged hilar nodes. This bronchus is long and narrow at the origin and its thin walls are completely surrounded by lymph nodes which drain the middle lobe, the right lower lobe and often the right upper lobe. This mechanical obstruction may also exist in any part of the pulmonary system. Calcified lymph nodes may partially or completely erode the bronchus and produce obstruction.

One cannot say which comes first, asthma and bronchospasm, or bronchiectasis, but the presence of bronchiectasis in an asthmatic is most important no matter how small it may be. Bronchospasm may cause bronchiectasis and conversely the bronchiectatic secretions may act as the initiating step in the production of attacks of bronchospasm. Thus, one often sees bronchiectasis and bronchospasm acting together in a vicious cycle.

Bronchiectasis is primarily a segmental disease, frequently bilateral, and may occupy any one of the 18 segments of the two lungs. The usual disease pattern includes involvement of the basal segments, the middle lobe or the lingula. Any asthmatic patient with symptoms of purulent sputum, hemoptysis, repeated respiratory infections or clinical findings of localized rales should be suspected of having bronchiectasis. There is little correlation between the severity of the symptoms and the number of segments involved with bronchiectasis. We have found that many patients with only middle lobe bronchiectasis may have symptoms much more disabling than those with more extensive bronchiectasis.

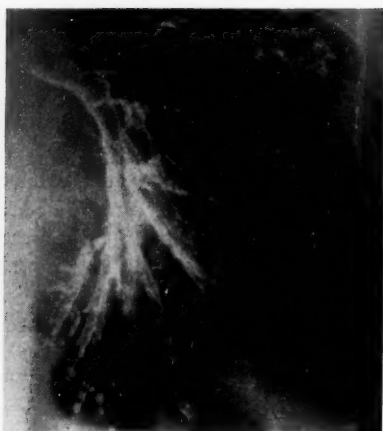


Fig. 1.—H. K. Cylindrical bronchiectasis of the left lower lobe.

All of these patients are bronchoscoped during their period of investigation. Often, it is necessary to do repeated bronchoscopies, sometimes under deep ether anesthesia. In addition to removing the thick, tenacious and adhesive secretions, at times, one may localize a lesion by observing bronchostenosis, obstruction due to extrinsic pressure, or origin of purulent secretion arising from bronchiectatic segments.

Accurate appraisal demands bronchography, although it is often difficult to perform and many times it becomes necessary to repeat it two or three times before a satisfactory pattern of the bronchial tree is obtained. These patients are questioned closely as to their drug sensitivities. They are carefully prepared for bronchography for several days with varied amounts of bronchodilators. Topical cocaine anesthesia has been used with good results. Bronchography is never attempted on a patient who is having moderate or serious respiratory distress. Even with adequate preparation, at times the patient may develop such severe bronchospasm that only the main stem bronchi are outlined with the contrast media. This examination must be repeated when the patient is free of bronchospasm.

Haphazard or inadequate preparation will seldom reveal the trigger mechanism, hidden or unsuspected bronchiectasis, in the asthmatic patient. DiRienzo⁴ has noted (1) the evident reduction in the caliber of the bronchial branches of the second, third and fourth order in one segment or zone and not in the entire lung, (2)

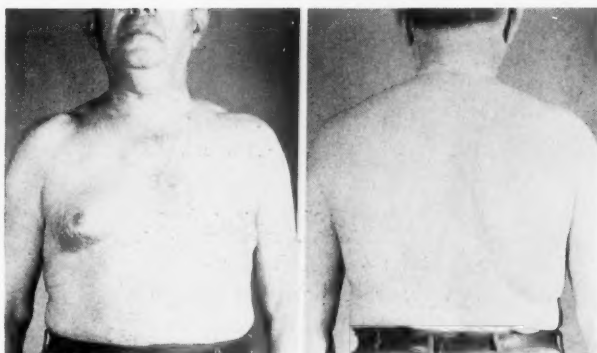


Fig. 2.—H. K. Postoperative photograph showing bilateral thoracotomy incisions.

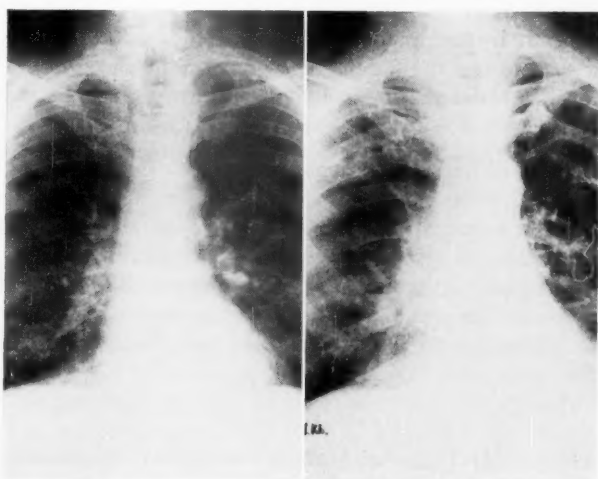


Fig. 3.—H. K. a. Pre-operative chest x-ray. b. Postoperative chest x-ray.

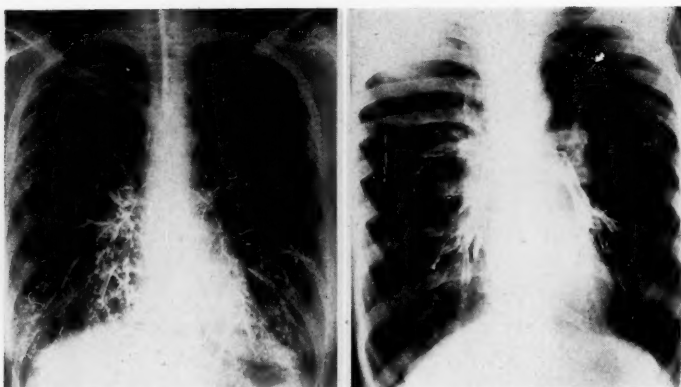


Fig. 4.—E. K. Incomplete filling of middle lobe.

Fig. 5.—H. L. Unsatisfactory bronchogram following inadequate preparation.

the absence of fine branching of the terminal bronchus in the infected segment, (3) the lack of foliage in this area, (4) the great delay in the progression of the contrast media in the abnormal segments, (5) the presence of numerous gas bubbles, and (6) the unchanged caliber of the infected bronchus during inspiration and expiration. It is most important to record changes and findings that are observed during the fluoroscopic guidance of the contrast media into the bronchial tree. This and the information from the x-ray films may enable one to locate the bronchiectasis.

Once the diagnosis of bronchiectasis has been made in a patient with bronchial asthma, the treatment is surgical unless the disease is so extensive it cannot be removed. Our experience clearly demonstrates that the end results in treating the bronchiectasis and bronchial asthma parallels the surgeon's ability to remove all of the diseased portions of the lung and to conserve all normal tissue. Segmental resection allows one to follow these two basic fundamental surgical principles.

CASE HISTORIES

CASE 1.—Mr. H. K., 63 years old, complained of chronic cough, purulent and occasionally blood-streaked sputum for 15 to 18 years. He gradually developed attacks of asthma over a period of 10 to 12 years. His previous therapy included adrenalin, aminophyllin, change in diet, oxygen, bronchoscopy and antibiotics. Bronchography re-

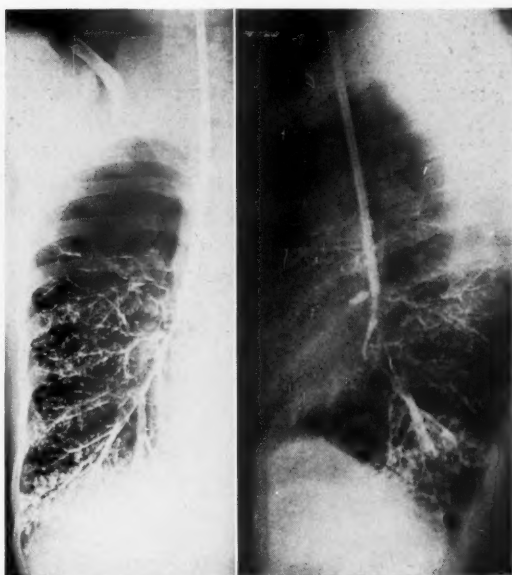


Fig. 6.—H. L. Satisfactory bronchogram demonstrating obstruction of middle lobe and anterior basal segmental bronchus.

vealed cylindrical bronchiectasis in the left lower lobe (Fig. 1). A left lower lobectomy was done in 1948 and excision of calcified lymph nodes about the middle lobe bronchus in May, 1951 (Fig. 3). He is free of cough, sputum, wheeze and hemoptysis and is able to work every day (Fig. 2).

CASE 2.—Miss E. K., a 32 year old nurse, complained of attacks of asthma of 13 years' duration. A few years later she began to develop repeated respiratory infection and was hospitalized several times for pneumonia. A 24 hour sputum specimen averaged 60 cc and occasionally was blood-streaked. She complained of rattling in the right lower chest and had pain in this area. Repeated allergic studies were of no benefit. Originally the bronchodilators helped but gradually became ineffective. Four hundred thirty bronchoscopies were done for aspiration of secretions and instillation of drugs. Bronchography in October, 1950 demonstrated incomplete filling of the middle lobe (Fig. 4). In December, 1950, a middle lobectomy was done and since then the patient has been free of symptoms and able to return to work.

CASE 3.—Mr. H. L., 15 years old, has had asthma since childhood. Originally the attacks were worse in the winter months, but during the past few years, there has been no real seasonal change in severity of attacks. There was no history of purulent sputum or hemoptysis. A bronchogram was attempted without adequate preparation (Fig. 5) and was totally unsatisfactory. One week later a repeat bronchogram revealed obstruction of the middle lobe bronchus and the anterior basal bronchus of the right lower lobe and bronchiectasis of the posterior basal segment of the left lower lobe (Fig. 6). Exploratory thoracotomy was carried out in November, 1951, the middle lobe was partially collapsed and nodulation was palpable throughout the lobe. The middle lobe bronchus was surrounded and compressed by enlarged lymph nodes. The anterior basal segment was found to be normal. A middle lobectomy was done and since then the patient has been markedly improved. It may be necessary to excise the abnormal segment of the left lower lobe.

Bronchiectasis had not been suspected in these three cases before our bronchography. Patient H. K. developed attacks of bronchospasm following several years of bronchiectatic symptoms. Patient E. K. developed definite bronchiectatic symptoms following onset of bronchial asthma. Patient H. L. did not have bronchiectatic symptoms but bronchoscopy and thoracotomy demonstrated definite pathology of the middle lobe.

RESULTS

During the past four years, 76 patients with severe bronchial asthma were treated at this clinic. Prior to our seeing these patients, bronchography had been done in only four patients. Of these, one bronchogram was satisfactory and this demonstrated bilateral basilar bronchiectasis. Bronchography was performed on the remaining 75 patients without bad effects and bronchiectasis was demonstrated in 27 patients. All were treated by exploratory thoracotomy and the abnormal segments were removed in 26 patients. Two patients with infectious complications were not improved with surgery, and in one patient, the bronchiectasis was too extensive for removal. Twenty of the 26 patients are markedly improved, working, and only seven of them require bronchodilator drugs.

CONCLUSION

Asthmatic patients apparently have a high incidence of bronchiectasis and this may be either a complication or an etiological factor in producing asthma. It should always be suspected in cases of intractable bronchospasm. Proper treatment of the bronchiectasis yields good results as therapy for the bronchospasm.

REFERENCES

1. Prickman, L. E., and Moersch, H. J.: Bronchostenosis Complicating Allergic and Infectious Asthma, *Ann. Int. Med.* 14:387, 1940.
2. Brock, R. C.: Post-tuberculous Broncho-stenosis and Bronchiectasis of the Middle Lobe, *Thorax* 5:5, 1950.
3. Graham, E. A., Burford, T. H., and Mayor, J. H.: Middle Lobe Syndrome, *Postgraduate Medicine* 4:29, 1948.
4. DiRienzo, S.: Radiologic Exploration of the Bronchus, Charles C. Thomas, Springfield, Illinois, p. 190, 1949.

DISCUSSIONS

DR. SHIRLEY H. BARON (San Francisco, Calif.): As the paper progressed, I wondered, from the cases presented, which was first, the horse or the cart? These cases appeared to fit a definite pattern of some primary infection followed by the development of asthma. I should like to ask Dr. Walker if his group of cases included some with primary asthma followed by secondary infection?

DR. FRANCIS W. DAVISON (Danville, Pa.): Dr. Walker shows excellent results and undeniably many bronchiectatic people with asthma do require lobectomy. I have had people who were asthmatic, who had minimal bronchiectasis, and responded very well to large doses of penicillin. By that I mean five, ten, or fifteen million units of fortified procaine penicillin per day for a week or ten days. We hear this loose term "antibiotic therapy." Too few people stop to specify the dose.

You will recall that not many years ago, subacute bacterial endocarditis was said to be totally incurable with penicillin, and the Penicillin Committee of the N. R. C. threw out penicillin as being useless treatment. Later Leo Loewe showed that penicillin could cure subacute bacterial endocarditis in a high percentage of cases if *bactericidal* doses, sometimes 40 million units daily, were used.

I think patients having infectious asthma and *minimal* bronchiectasis deserve a trial with *bactericidal* doses of penicillin *plus* surgical treatment of any coexisting sinus infection. If this fails, lobectomy as demonstrated by Drs. Overholt and Walker may be the answer. Certainly lobectomy is necessary when bronchiectasis is extensive.

DR. EDGAR P. CARDWELL (Newark, N. J.): I would like to ask if there has been any subsequent difficulty from nasal infection in those cases, or whether they were cases entirely free of nasal trouble? So many cases I see have had accompanying nasal infection. I have had some success with asthma in treating the nasal infection alone in some severe cases, without the help of removal of the infected pulmonary segment, or where more general pulmonary changes were present.

DR. JAMES H. WALKER (Brookline, Mass.) In answer to the first question, the only selection we made of these patients was that they were all medical failures. That makes it a very select group because most of the patients with bronchial asthma are going to be controlled by broncho-dilators, by allergic measures, or antibiotics. Approximately half of this group had had severe infection prior to their attacks of bronchospasm. In the slides shown there was a preponderance of this type because they were good demonstration material, but it was about half and half. We believe that patients with bronchial asthma can develop bronchiectasis just as patients with bronchiectasis can develop bronchial asthma.

Some of our patients have had nasal surgery and sinus surgery prior to coming to us. We did not make any special effort to have nasal and sinus studies because they had all been investigated very carefully prior to coming to us. Many of them were referred by allergists.

FAT IN THE TRACHEO-BRONCHIAL TREE WITH REPORT
OF A CASE OF TRUE LIPOMA OF THE BRONCHUS

ALEXANDER H. BEATON, CAPT. M. C.

AND

CLYDE A. HEATLY, M.D.

ROCHESTER, N. Y.

Bronchial lipomas are relatively unusual tumors and their presence as well as that of fat in other bronchial lesions has led to considerable speculation about the origin of fat in this location. The single case of a bronchial lipoma which is presented stimulated our interest in this problem.

THEORIES OF ORIGIN

The origin of fatty tumors in any portion of the body is not clearly established. We know that fat is a product of the embryonal mesoderm, and that this mesenchymal tissue is the forerunner of bone, cartilage and connective tissue. Early in the development of the embryo differentiation of cell types occurs, at which time certain cells become lipoblasts. The cytoplasm of these cells gradually elaborates fat droplets which coalesce and form the large fat globule giving the cell its characteristic appearance. Groups of the immature cells cluster together and are usually found in association with blood vessels and connective tissue elements. These are probably the forerunners of larger fat lobules in adult life. Although tumors may arise from immature as well as adult fat cells, it is impossible to distinguish the origin of these lipomas in the adult. Any of the elements from early mesenchyme to the mature fat cell may be the source. In order to explain the presence of fat in lung tumors, many rather elaborate hypotheses have been put forth. Some authors feel that it might be the result of metaplasia of connective tissue elements; others that embryonal arrests are responsible. It has also been suggested that fat cells are carried to the periphery by the expanding elements of the lung as it develops. All of these imply that fat is an abnormal constituent of the tracheobronchial tree and must have

From the Department of Surgery, Division of Otolaryngology, University of Rochester School of Medicine and Dentistry.

a rather involved explanation for its presence. We do not feel that such is the case and wish to emphasize and further corroborate a simpler explanation as proposed by Watts¹ that fat should not be considered an unusual part of bronchial tumors, since it exists as a normal tissue component in the submucosa of the bronchial wall.

MICROSCOPIC STUDIES

To do this, a series of ten lungs obtained at autopsy were examined microscopically. The age of the patients varied from early childhood to the older age groups. The only selectivity employed was an attempt to use specimens in which the local pathology would not interfere with our examinations. Sections were taken at four different levels beginning at the lower trachea and extending out to the peripheral limits of the cartilaginous segments. These sections were stained by the ordinary hematoxylin-eosin method since we found that fat could be easily identified in this manner without employing more complicated staining procedures (Figs. 1, 2, 3, 4). However, for more striking contrast in demonstrating the location of this fat, several sections were stained with Sudan IV. This results in a heavy accumulation of dye in the regions previously occupied by fat as is clearly seen in the illustration (Fig. 5). In all, some 50 slides were examined and fat was found in the submucosal tissues of the tracheobronchial tree in every instance. It extended peripherally to the limits of cartilage and was always in close association with it. The more proximal specimens contained larger quantities of fat which were located primarily in the connective tissue interspaces and external to the cartilage. As more distal sections were obtained, the location became more variable, appearing around the glands and blood vessels as well as between the cartilaginous segments. However, both submucosal and periglandular fat was demonstrable at every level. It does not seem necessary, therefore, to postulate any embryonal aberration to account for the occurrence of fatty tumors in these structures since fat is present as a normal cell unit and may become involved in cellular overgrowth, as it does in other areas of the body, to produce a characteristic lipoma. A recent corollary to this work was demonstrated by Nuessle.² Working with a substance intimately related to the tracheo-bronchial tree and its mucous membrane, and using careful controls to eliminate contamination by ingestion, he has proved rather conclusively that fat is a normal constituent of bronchial sputum. It is of interest to note that fat is found in the variety of bronchial tumors. In the series of 40 cases of chondromas reviewed by Hickey and Simpson,³ 50 per cent of the tumors contained fat. Adenomas often contain variable quantities of adipose

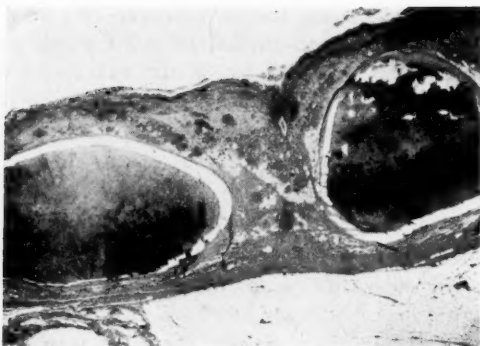


Fig. 1.—Vacuolated fat cells in the inter-cartilaginous tissue of the trachea (Mag. X 25).

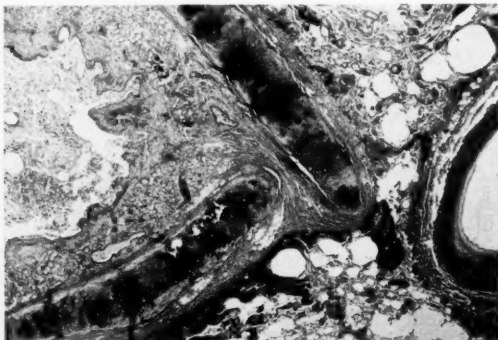


Fig. 2.—Fat both external and internal to the cartilage of a large bronchus (Mag. X 25).

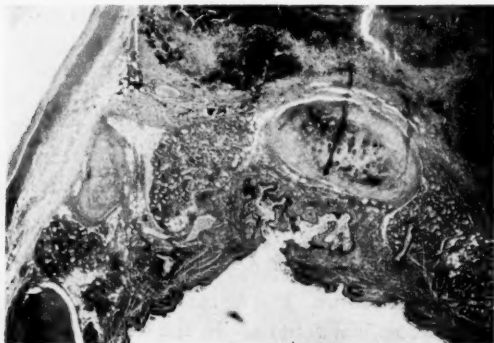


Fig. 3.—Periglandular fat in the region of a peripheral bronchus (Mag. X 25).

tissue. In the earlier literature tumors, reported by Rokitansky and Laboulbene were considered lipomas because of their fat content although further study in recent years has pointed to other cell elements as being more important in the tumor structure. The fibrolipomas of Meyerson,⁴ Wessler and Rabin,⁵ Jackson and Jackson,⁶ and McGlade⁷ of course fit into this group. The hamartomas of both Feller⁸ and Honig⁹ contained lipoid tissue and two thirds of the 23 cases of similar tumors reported by McDonald, Harrington and Claggett¹⁰ contained fatty tissue elements. In addition to these bronchial tumors, a tracheal fibroma containing fat cells was reported by Zamora.¹¹ All of these cases serve to emphasize our hypothesis that fat is a normal constituent of the tracheobronchial tree and as such is frequently involved in pulmonary pathology.

REVIEW OF REPORTED CASES

In reviewing the cases of lipoma previously reported, an endeavor has been made to include only those tumors which in the majority opinion were unquestionably true lipomas. This necessitated excluding fibrolipomas, hamartomas and similar tumors in which fat was a component but not the primary cell type. Of the 11 cases of true bronchial lipoma reported in the literature up until this time, seven were diagnosed and treated bronchoscopically, two were treated by thoracic surgery and two were reported as findings at autopsy. Variations in pathological diagnosis are great even among the pathologists of a single institution so that without adequate opportunity to evaluate the microscopic specimens of each case, one must draw conclusions from the authors' nomenclature or description.

The first undisputed case of lipoma was reported by Kernan¹² in 1927. This occurred in a 50 year old man with a history of cough for five years. The tumor was located to the right of the tracheal bifurcation and was attached by a broad pedicle to the lateral wall. Removal was accomplished by severing the pedicle with biting forceps.

Meyerson⁴ included in his paper two cases of lipoma found at autopsy. One was located in the left lower lobe bronchus and the other in the right middle lobe bronchus but no further details were given.

In a comprehensive report on benign tumors of the tracheobronchial tree published in 1932, Jackson and Jackson⁶ reported a lipoma in a 64 year old female. This was found bronchoscopically in the left bronchial orifice. It was removed successfully with forceps and no pulmonary symptoms were evident after a three-year follow-up period.



Fig. 4.—The arrows indicate the persistence of a small quantity of fat associated with the cartilage in bronchi of this minute diameter (Mag. X 25).



Fig. 5.—Blackened areas indicate fatty tissue stained by Sudan IV in a stem bronchus (Mag. X 25).



Fig. 6.—Section of lipoma removed from left main bronchus (Mag. X 25).

No further cases were reported for 10 years until Vinson and Pembleton¹³ reported a lipoma on the anterior wall of the left main bronchus in a 29 year old Negro female. This tumor had a wide base and suppuration distal to it seemed to render a pneumonectomy imperative. The operation was refused, however, and coagulation therapy was initiated but never completed because of the patient's failure to return.

A lipoma reported by Watts¹ was found at operation in the main bronchus of the left upper lobe of a 50 year old man. It had not been possible to visualize this bronchoscopically prior to thoracotomy.

Bronchoscopic diagnosis and treatment of a lipoma in the left stem bronchus of a 61 year old man was successfully carried out by Whalen¹⁴ in 1946. The base of the tumor was broad and firm. Removal was somewhat difficult and was followed by mediastinitis from which the patient rapidly recovered under antibiotic therapy.

In 1949 Lell¹⁵ reported a case of lipoma of the right main bronchus in a 40 year old white male, which was successfully treated by multiple bronchoscopic avulsions.

A true lipoma of the left upper lobe bronchus in a 52 year old white female was found at operation by Carlisle¹⁶ and reported in 1951. The tumor had been visualized prior to lobectomy by bronchoscopic telescopy.

Two cases were reported recently by Som,¹⁷ both males, ages 55 and 59. In the former case, the tumor was in the right main bronchus with its pedicle in the lower lobe bronchus. The latter had its origin at the entrance to the right upper lobe orifice. In both instances diagnosis was made bronchoscopically and treatment was carried out successfully by forceps evulsion.

CASE REPORT

The following is a case which was diagnosed and treated bronchoscopically with satisfactory outcome.

L. S., a 36 year old negro, entered the Strong Memorial Hospital on October 31, 1949 complaining of chills and left sided pleuritic pain. For two months prior to admission he had been troubled by a moderately productive cough. Roentgenograms obtained by the patient two weeks previously at another institution demonstrated increased markings in the lower left lung field. Repeat studies were scheduled but this acute episode interrupted the plan.

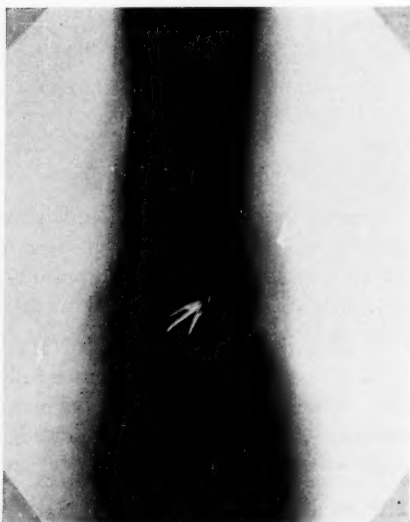


Fig. 7.—Tomogram showing location of the tumor in the left main bronchus as outlined by the arrows.

His physical findings were typical of left lower lobe pneumonia and roentgenograms confirmed the diagnosis. The organism cultured from the sputum was pneumonococcus type 2 and the illness responded promptly to penicillin so that within three days the temperature fell from 40.3°C to normal. The antibiotics were discontinued on the 13th day, and he was discharged on the 17th hospital day. His slightly prolonged course was accounted for by rather resistant left sided chest pain and excessive sputum. Both of these symptoms had largely cleared on discharge. Serial roentgenograms demonstrated a regression of his pulmonary disease. Additional sputum studies for acid fast bacilli were negative.

For six months he was followed in the Out Patient Department by the Medical Service. Repeated chest films showed no recurrence of his parenchymal pathology, but despite this objective evidence of improvement, he continued to have cough and left chest pain. Therefore, on April 27, 1950, bronchoscopy was performed. A smooth, rather pale pink mass about one and one-half cm in diameter was visualized approximately two cm from the carina in the left main bronchus. It almost completely obstructed the lumen of the

bronchus and seemed to be attached by a rather broad base to the floor and lateral wall. Several pieces of tissue were removed with minimal bleeding, making it possible to pass suction beyond the mass. No purulent material was obtained distal to this area. An attempt was made to remove the tumor intact through the bronchoscope, but because of its unusually broad base, this was not feasible. Microscopic examination of the tissue appeared characteristic of a lipoma showing a fine collagenous reticular stroma surrounding numerous fat vacuolated spaces, (Fig. 6) Tomograms (Fig. 7) demonstrated no peribronchial extension and therefore treatment was undertaken endoscopically, using an insulated Jackson bronchoscope and the Bovie unit to coagulate the base of the tumor. Further studies of the secretions including smears, cultures, guinea pig inoculations for acid fast bacilli and repeated cytological examinations were all negative.

During the period of treatment, the patient was followed by serial roentgenograms and repeated biopsy of the site of the tumor. The tumor decreased progressively in size so that by December 1950 all that remained was a slight irregularity in the floor of the left main bronchus at the original location of the lipoma. The 8 mm 'scope could be passed distally to this and no abnormality was noted. The patient has been followed by bronchoscopic examination every two months since final coagulation, and as of the last examination, no evidence of recurrence had manifested itself. Complete pulmonary evaluation, including ventilation studies, cardiac catheterization, balistocardiography and electrocardiography has been done. Except for slight diminution of vital capacity and maximum breathing capacity, all findings were normal. Repeated bronchography has failed to outline clearly the basilar segments of the left lower lobe although fluoroscopically there is indication of continued elasticity of these bronchi. It is our intention to continue a careful follow-up of this man. At the present time, bronchoscopic treatment of the tumor appears to have been satisfactory and no further surgery seems indicated.

SUMMARY

The probable etiology of bronchial lipomas is suggested by a series of sections of the tracheobronchial tree at different levels which demonstrate the presence of fat as a normal submucosal constituent. A brief review of the literature of lipomas of the tracheo-bronchial tree is presented. A single case diagnosed and treated endoscopically is added to the 11 cases of true lipoma of the bronchi which have been recorded in the literature to date.

REFERENCES

1. Watts, C. F., Claggett, O. T., and McDonald, J. R.: Lipoma of the Bronchus: Discussion of the Benign Neoplasms and Report of a Case of Endobronchial Lipoma, *J. Thoracic Surg.* 15:132-144 (Apr.) 1946.
2. Nuessle, W. F.: Significance of Fat in the Sputum, *Am. J. Cl. Path.* 21: 430-435 (May) 1951.
3. Hickey, P. M., and Simpson, W. M.: Primary Chondroma of the Lung: A Roentgenological and Pathological Study Based upon an Analysis of Forty Cases, with a Report of Two New Cases, *Acta Radiol.* 5:475-500, 1926.
4. Meyerson, M. C.: Benign Neoplasms of the Bronchus: Report of a Case of Fibrolipoma of the Left Main Bronchus Removed Through the Bronchoscope, *Am. J. M. Sc.* 176:720-726 (Nov. 19) 1932.
5. Wessler, H., and Rabin, C. B.: Benign Tumors of the Bronchus, *Am. J. M. Sc.* 183:164-180 (Feb.) 1932.
6. Jackson, C., and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi with Special Reference to Tumor-like Formations of Inflammatory Origin, *J. A. M. A.* 99:1747-1754 (Nov. 19) 1932.
7. McGlade, T. H.: Fibrolipoma of the Bronchus: Report of a Case, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 48:240-243 (Mar.) 1939.
8. Feller, A.: Über Ein lipomähnliches Harmartom der Lunge, *Virchow's Arch. f. path. Anat.* 236:470-480, 1922.
9. Honig, A.: Ein lipomartiges Gebilde des linken Stammbronchus, *Monatsschr. Ohrenh.* 68:155-167 (Feb.) 1934.
10. McDonald, J. R., Harrington, S. W., and Claggett, O. T.: Harmartoma (Often called chondroma) of the Lung, *J. Thoracic Surg.* 14:128-143 (Apr.) 1945.
11. Zamora, A. M.: Two Benign Growths Removed by Bronchoscopy, *J. Laryng, and Otol.* 46:829-833 (Dec.) 1931.
12. Kernan, J. D.: Three Unusual Endoscopic Cases, *Laryngoscope* 37:62-64 (Jan.) 1927.
13. Vinson, P. P., and Pembleton, W. E.: Lipoma of the Left Main Bronchus, *Arch. Otolaryng.* 35:868-870 (June) 1942.
14. Whalen, E. J.: Lipoma of the Bronchus, *Ann. Otol. Rhin. and Laryng.* 56:811-818 (Sept.) 1947.
15. Lell, W. A.: Report of a Case of Fibrolipoma of Right Main Bronchus; Bronchoscopic Removal, *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY* 58:1124-1134 (Dec.) 1949.
16. Carlisle, J. C., Leary, W. V., and McDonald, J. R.: Endobronchial Lipoma: Report of Case, *Proc. of Staff Meet., Mayo Clinic* 26:103-106 (Mar. 14) 1951.
17. Som, M. L., and Fuerstein, S. S.: Endoscopic Removal of Lipoma of the Bronchus, *Arch. Otolaryng.* 54:341-346 (Oct.) 1951.

DISCUSSIONS

DR. CLYDE A. HEATLY (Rochester, N. Y.): This case is not presented as a medical curiosity, but as an example of a relatively rare tumor which is becoming increasingly familiar.

It is significant, I think, that during the last six years since Dr. Whalen presented his case of true lipoma, six cases have been found bronchoscopically and treated successfully bronchoscopically, meaning that 50 per cent of the re-

ported cases have been found and treated successfully through the bronchoscope. This is not so much a tribute to the bronchoscopist as it is to the increasing awareness of the general physician to the implication of symptoms of bronchial stenosis which have been so well taught by the senior members of this Association.

All of these cases show similar symptoms of repeated attacks of so-called virus pneumonias, unresolved pneumonias, x-ray evidence of segmental atelectasis, unexplained wheezing—a variety of symptoms which indicate not only this type of tumor but many other types of tumor, as you know.

Lipomas, of course, are found not only in the tracheobronchial tree but in the chest wall, the pleura, and the mediastinum. The presence and relation of fat to the tracheobronchial tree, of course, is well known to pathologists. So far as I could determine, it is less well known to members of our group. It occurred to me that it might be of interest to show you once again the distribution of fat in relation to the tracheobronchial tree, the more so because so many benign tumors contain fat elements.

DR. WILLIAM A. LELL (Philadelphia, Pa.): In the last six months, we came across another case of so-called lipoma, and again this patient presented the picture, not of a benign lesion, but of a possible malignant process. On bronchoscopic examination, however, we saw a large mass filling the entire lumen of the bronchus.

As you know, lipomas do not infiltrate but fill space, and that is one of the reasons why these tumors may not produce any symptoms over a long period of time until they obstruct.

We followed the case regularly at intervals of three months, then six months, and now a year. There has been no evidence of recurrence except for the thickened appearance of the mucosal surface at the site of origin.

DR. CHARLES M. NORRIS (Philadelphia, Pa.): I would like to mention briefly a similar case observed on the service of Dr. Chevalier L. Jackson at Temple University Hospital. This case may not be strictly comparable, in that the tumor contained some fibrous element. One reason for describing it, however, is that we obtained endobronchial photographs of the lesion, which might be of interest. [Two slides and a film were shown.]

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 3, 1952

THE PRESIDENT, DR. ARTHUR J. COOMBS, IN THE CHAIR

The Otolaryngologist in a Hearing Conservation Program

RICHARD E. MARCUS, M.D.

The Chicago Hearing Conservation Program is the result of co-operation between representatives from the Chicago Medical Society, the Chicago Laryngological and Otological Society, the Board of Education, the Chicago Hearing Society and citizens' school groups. Trained audiometrists follow a pre-determined pattern of testing. Otologic screening clinics have been established for children found to have a hearing loss of 30 decibels or more in more than one frequency in either ear. Tentative diagnoses are made in the screening clinics; adequate follow-up is made to ensure the therapeutic and rehabilitative management in private or public hands. Educational placement of children is made on an individual basis, depending on the type of hearing loss, prognosis and duration.

From September, 1950, to February, 1952, 49,574 children were screened audiometrically. Of these 2.5% were found to have a hearing loss of 30 decibels or more, in more than one frequency in either ear. Of these, 85% appeared at the otologic screening clinics, and 84% were found to have a hearing loss, in 63% of whom this had not been previously known. On the six months recheck of 304 children for whom medical care was recommended and carried out, 58% showed improvement, 34% showed no improvement, and 8% were worse. Where medical care was recommended but not carried out, 32% improved, 56% showed no improvement and 10% were worse. In children in whom no medical care was recommended, 58% showed improvement, 35% showed no improvement and 7% were worse. In those children referred by teachers from grades others than the ones screened, and ungraded classes, 5.5% had actual loss.

It is noteworthy that, where medical care was recommended and carried out, six out of ten children showed improvement. In this same group, where medical care had not been carried out, only three of ten children showed improvement. Statistically, therefore, there is evidence that hearing loss in children can be improved following accurate diagnosis and specific treatment. Further studies are indicated and are in progress.

DISCUSSION

DR. KENNETH NOLAN (Director School Health Service): Much of the difficulty in the effort to secure optimal educational placement has been due to the fact that the Board of Education and organized medicine, by and large, have not had the opportunity to come together to consider some of these problems. It is heartening that this Society has furnished a very excellent example. This has already encouraged several other similar groups to supply consultation and advice on various matters.

The responsibility of the health of the child rests with the parents. The responsibility of the Board of Education is that tax funds earmarked for educational purposes is used; therefore any project undertaken must be justified educationally. It is justified when it protects the initial investment. It costs \$256 a year to keep a child in an elementary school classroom. When he fails a year in school because of poor hearing, it is obviously good economy to have that information beforehand and secure better adjustment and placement for the child. Special school placement has been done in the past almost entirely devoid of any medical consultation. At this time 60 or more speech correctionists are undertaking correction in speech in many children in whom it has just been discovered that a hearing loss is present. In the rooms for the retarded, twice as many mentally handicapped children are found to have a previously unknown hearing loss, as children in the regular elementary classrooms.

The otologist can be of great aid in advising on acoustics in newly built schools; auditory considerations are often poor. In one school where a large number of children were seen who, on the basis of initial group tests were apparently not meeting requirements, it was found that the school's proximity to the airport interfered seriously with the audiometry tests.

Much remains to be done in the program, and further planning will be carried on only with prior consultation and advice of organized medicine.

DR. JOHN L. REICHERT: I have had the opportunity of watching the work of Dr. Marcus and Dr. Nolan from its inception. A planning Committee was organized which consisted of all volunteer and official agencies interested in school health. It has been planned as a very broad-base, city-wide program extending far into the future. Much credit must be given to the Chicago Hearing Society, which underwrote the program before official funds could be obtained. Initially a case finding program was started which went on to the stage of making an exact diagnosis and that, as Dr. Marcus said, was done through the otologic screening clinics. In these clinics there is no therapy; when the diagnosis is made the case is sent to the practicing physician in his office or in the dispensary. Organized medicine has accepted the principle that in community health programs mass diagnosis on a public health basis is justified, but has strictly adhered to the principle that therapy is definitely the province of the practicing physician.

Here we have a solid program which consists of case finding, diagnosis of hearing loss and, finally, rehabilitation. Rehabilitation consists not only in giving what remedial care is possible, but in adjusting the child to his physical handicap, which is most important.

In one school in the city, six children were found who had a remedial hearing loss. Those six children represented a total of ten semesters of repeated grades because of poor hearing. When the hearing loss was remedied, their work improved and they will not repeat another semester. Five years of repeats represents, as Dr. Nolan indicated, about \$1,200 loss of taxpayers' money. Since there are more than 400 schools, that would mean approximately half a million dollars annual loss before this program went into effect. In other words, the program is more than maintaining itself.

As has been shown, two-thirds of the patients with hearing loss so far uncovered were in school for a long period without the impairment being discovered. Teachers at present cannot be relied upon for screening purposes. The physician has a broad responsibility to the community, and such a program as this enables us to fulfill that responsibility.

DR. ARTHUR J. COOMBS: I should like to ask what the otologist should do, assuming that he is the first to discover impaired hearing in a child. Where should he send the child, and where can the program best be carried out?

DR. KENNETH NOLAN: The physician can apply to the Department of Special Education for placement for the child. If lip

reading or other special facilities are required, there are rooms incorporated in the regular schools in various sections of the city. There are also sight-saving classes in rooms in various schools. By the use of a hearing aid, in some instances a special class may not be needed.

Otogenic Intracranial Complications in the Antibiotic Era

MARVIN TAMARI, M.D.

AND

ROBERT HENNER, M.D.

(Abstract)

In the management of acute otitis media penicillin has proven to be the drug of choice. In those cases in which penicillin is not immediately effective there is a tendency to haphazardly prescribe an array of antibiotics. The procedure of administering multiple antibiotic agents at the same time and without bacteriologic and sensitivity studies has proven of little value and, indeed, complicates the clinical picture. One should be especially alert for the existence of unsuspected complications.

The cases presented were selected from many problems of otogenic seen at the Illinois Eye and Ear Infirmary during the era of antibiotic therapy. They serve to dissipate the illusion that mastoid disease is always prevented or cured by antibiotics, and to illustrate the need for adequate surgery when indicated. It has been the experience that when surgery is indicated, antibiotics have failed to prevent complications or effect a cure. If surgery is necessary, it should be thorough, meticulous and complete, as the standards of pre-antibiotic days indicated.

DISCUSSION

DR. ROBERT HENNER: I would like to point out that in addition to emphasizing the lack of osteoclasia in these cases, a recent article by Mock in the Journal of the American Medical Association showed that penicillin favored thrombosis. Dr. Tamari has demonstrated the extensive thrombosis along the dural plate. This brings up the question whether these changes are in fact not a sequel to the use of antibiotics. We intend to make further studies along this line.

Certainly we all hope that future studies in bacteriologic and chemotherapeutic agents will present us with the proper antibiotic

for every infection. However, this paper was presented to show that undue reliance upon these agents, and the ignoring of the sound surgical principles of the pre-antibiotic days, may cause serious difficulty in the handling of patients.

Meeting of Monday April 7, 1952

THE PRESIDENT, DR. ARTHUR J. COOMBS, IN THE CHAIR
**Thyrotomy Approach for Arytenoidectomy in Bilateral Abductor
Paralysis of the Vocal Cords**

SAMUEL J. PEARLMAN, M.D.

CHICAGO, ILL.

AND

EDGAR W. KILLIAN, M.D.

LOGANSPOUT, IND.

The authors have reviewed a number of procedures designed to provide adequate breathing space and eliminate the tracheotomy tube in bilateral abductor paralysis, and describe a method of unilateral arytenoidectomy through a thyrotomy which was performed in four cases. The operations were successful in that all patients have an adequate airway and a useful voice. A comprehensive bibliography is included.

**Nasal Implants in Children and in Adults, with Preliminary Note
On the Use of Ox Cartilage**

MAURICE H. COTTLE, M.D.

THOMAS J. QUILTY, M.D.

AND

RICHARD A. BUCKINGHAM, M.D.

CHICAGO, ILL.

During the past five years a large variety of rhinoplastic procedures have been used in the effort to correlate surgery of the external pyramid with intranasal operations in the restoration of nasal

function. Among these is utilization of implants of inorganic and organic materials, autogenous and isogenous bone and cartilage. In addition to restoration of contour and establishment of support, functional indications such as repair of the nasal roof, restoration of the hard nasal septum, narrowing of excessively widened nasal chambers and repair of deformities of the nasal lobule are now considered.

The paper discusses the use of bone, fresh and preserved, autogenous and preserved cadaver cartilage, and ox cartilage. No one material is as yet universally adequate; autogenous cancellous bone when indicated is the best graft material available. The authors recommend the use of implants in children, in repair of destroyed septal cartilage following abscess of hematoma, in order to prevent later deformity.

Abstracts of Current Articles

EAR

Inner Ear in Presbycusis.

Saxon, A.: *Acta Oto-Laryngologica* 41:213-227, 1952.

While presbycusis may be due to arterio-sclerotic lesions in the central nervous system, senile atrophy of the spiral ganglion possibly accompanied by angio-sclerotic degeneration of the inner ear usually is present and probably is the most significant factor producing the hearing loss.

HILL.

Recording of Objective Tinnitus.

Engstrom, H., and Graf, W.: *Acta Oto-Laryngologica* 41:228-234, 1952.

Objective tinnitus may be of vascular origin and arise in the mandibular joint, the soft palate, or the muscles of the middle ear. The authors describe the use of a calibrated phono-cardiograph for the recording of audible tinnitus.

HILL.

Occupational Injuries of the Ear.

Ruedi, L.: *Acta Oto-Laryngologica* 41:118-138, 1952.

Aero-otitis media cannot be fully explained by the difference in air pressure between the middle ear and the outside world, inasmuch as the symptoms may persist even if adjustment of pressure is established early. Animal experiments have shown the same picture may be produced by either diminution or excess of oxygen. Maintenance of normal range of oxygen pressure may be fully as important prophylactically as an open eustachian tube.

Acoustic trauma may be due either to noise or explosive blast. Following investigation both on military personnel and on experimental animals it has been shown that ears may be completely protected from all kinds of acoustic trauma without noticeable impairment of hearing by means of "ear-defenders," utilizing the principle of sound filtration.

HILL.

Finnish Speech Audiometry.

Palva, T.: *Acta Oto-Laryngologica*, Suppl. 101, 1952.

This comprehensive and well written treatise of 125 pages includes a review of speech hearing tests with an evaluation of pure tone audiograms in terms of speech hearing. The author describes his own investigations aimed at developing a method of speech audiometry adapted for the Finnish language. The importance of speech audiometry is stressed, especially in prescribing hearing aids, in selecting cases suitable for fenestration, evaluating the results, and in determining the loudness function where both ears show the same air conduction loss. The author concludes that pure tone audiometry and speech tests supplement each other and both should be employed in order to achieve a more complete understanding of the type and degree of hearing loss than can be obtained by either method alone.

HILL.

Evaluation of Hearing in Young Children.

Hardy, W. G., and Bordly, J. E.: *Acta Oto-Laryngologica* 40:346-360, 1951-52.

This is a comprehensive description of galvanic skin-resistance audiometry as developed at Johns Hopkins. The technique is exacting and not adaptable to the clinician's office. It is only one step in a thorough study encompassing exposure of the child to many acoustic stimuli. When the rudimentary clinical tests are inconclusive, reference to a center equipped and staffed to carry out all diagnostic methods is advisable. At the present time the authors feel that skin resistance audiometry is the only method which meets all the criteria applicable to diagnostic testing in infants and very young children.

HILL.

Medical and Psychological Consideration of a Hearing Aid.

Glorig, A.: *Acta Oto-Laryngologica* 40:370-375, 1951-52.

Cases with a pure conduction loss should benefit from the use of a hearing aid. The problem is with both the pure preception, or the mixed type with predominant nerve loss. As the average gain for speech with a hearing aid is 40-45 db, losses of greater than 65 db cannot be compensated for satisfactorily, with rare exception. While most cases with losses of less than 30 db do not need a hearing aid, there are those with this loss or losses, the so-called "lazy listeners," who are definitely benefitted by amplification.

Other considerations are the length of time the loss has been present and the confusion of ambient background noise, the presence of recruitment, the age, and economic, and social background of the patient. Perhaps of greatest importance is the availability of auditory training which may be of great value in helping the patient use his aid most effectively.

Except for cases with active middle and external ear disease or distorted external meati, the air conductive type of aid is preferable, despite the bone conduction curve.

The importance of a well-fitted, individually tailored ear-piece is obvious. The author has developed a soft plastic ear insert which produces a nearly perfect acoustic seal that is more comfortable than those now available.

The use of a hearing aid may seem to give improvement in hearing probably through psychophysiological coordination. Rarely damage may be done when the resonant peaks of microphone and receiver combine to produce 135 db.

The attitude of the patient toward his deafness demands consideration and may indicate psychotherapy, as a part of the program of auditory training.

HILL.

The Use of Extreme Limitation for the Dynamic Equalization of Vowels and Consonants in Hearing Aids.

Edgardh, B. H.: Acta Oto-Laryngologica 40:376-382, 1951-52.

One difficulty in attaining satisfactory performance from a hearing aid, particularly in cases with nerve impairment, is due to the diminution of the dynamic range between thresholds of hearing and pain. When sufficient amplification is given to allow consonants to be heard, the vowels will be too loud. Experiments aimed at constituting a spectrograph for dynamic-acoustic analysis of spoken sounds have suggested the possibility of solving this problem of dynamic vowel-consonant equalization.

This is a highly technical paper and the author frankly states that a great deal of further research is necessary before determining whether it will be practical to apply this principle to the hearing aid.

HILL.

Principles Involved in Selecting a Hearing Aid.

Glorig, A.: *Acta Oto-Laryngologica* 41:49-57, 1952.

The hearing aid is simply an audio amplification system modified by its necessary size. It converts sound into electrical energy, amplifies it 100,000,000 times and reconverts it to physical energy. This process may result in changes in frequency response, amplification, distortion, and extraneous noise. Control of amplification in relation to frequency is difficult to achieve.

Conduction deafness is rather easily corrected by a hearing aid but with perception deafness discrimination presents difficulties so that auditory training is important.

As a result of the experiences at the Army Audiological Center the author feels that a highly discriminative selection procedure is unnecessary in the conductive type as well as in 80% of perceptive cases, although in the latter these may have some psychological value. With any of the standard hearing aids the most important procedure to insure the best results is an adequate training program.

HILL.

NOSE**Fibromas and Angiofibromas of the Nasopharynx.**

Alcaino, Alfredo: *Rev. De Otorrinolaryng.* 12:2 (Sept.) 42, 1952.

This author proposes a new surgical approach for the elimination of fibromas and angiofibromas of the nasopharynx.

He presents evidence that these growths originate within the nasal cavity and extend into the nasopharynx which affords space for the expansion of the tumor.

His incision begins in the nasal vestibule at the upper anterior edge of the quadrangular cartilage and is carried down to the floor and beyond the midline. The mucosa and perichondrium are separated from the septum on both sides extensively as in submucous resection. The cartilage, perpendicular plate, and the vomer are removed as far back as possible. Any bony tissue which remains is separated from the floor so that the septum can be widely displaced laterally toward the healthy side. Both lower turbinates are fractured and pushed against the lateral nasal walls.

According to this author the procedure gives sufficient access to the nasopharynx and the nasal cavity for removal of the tumor.

He states that visibility is increased and thus hemorrhage is more easily controlled by this approach.

Six cases are reported in which the operation was completely satisfactory.

HIGBEE.

BRONCHI

Sudden Death From Bronchial Asthma Following Injection of Pyromen.

Walton, C. H., and Elliott, G. B.: *The J. of Allergy* 23:322-326 (July) 1952.

The authors report a case of sudden and unexpected death from asthma following the intravenous use of Pyromen. This patient had an intrinsic type of asthma starting in pregnancy which became very severe after a nasal polypectomy and double antrum operation. In the three and a half years she was under treatment she had recurrent exacerbations and remissions and was treated symptomatically. Two courses of ACTH produced remissions of asthma for a few weeks and restoration of psychological stability. Her fourth admission to hospital was characterized by a less severe asthma than on previous occasions. Therapy with Pyromen produced no beneficial results of any kind although administered for eight consecutive days. Following her last injection there developed a rapidly increased bronchial obstruction and death in 85 minutes.

The authors state that asthmatic patients of this type have a notoriously poor prognosis and often require very little to upset their delicately balanced respiratory state. They cannot be positive that the use of Pyromen was causally related to the patient's death but they do say with certainty that Pyromen had no demonstrable beneficial result.

VAN ALYEA.

ESOPHAGUS

On the Treatment of Corrosive Lesions in the Esophagus. An Experimental Study.

Krey, H.: *Acta Oto-Laryngologica*, Suppl. 102, 1952.

While a review of the literature indicates that most writers favor the institution of treatment before scar-tissue formation there is considerable difference of opinion as to the best method. Endeavoring to answer this problem the author carried out experimental corrosion

on 143 rabbits. This was followed by x-ray, histological, and bacteriological studies. One group of animals received no treatment following corrosion and served as the control. The rest of the rabbits were divided into three groups, one being treated with sulfonamides, one with indwelling tubes and sulfonamides and antibiotics, and the remaining with gastrostomy and sulfonamides and antibiotics.

The experiments show that secondary infection plays an important part in the development of stricture and that epithelization is hastened by the use of sulfonamides and antibiotics. If treatment is combined with an indwelling tube inflammatory changes are more widespread and stricture formation is increased. On the other hand the best results are obtained if the esophagus is put at rest by gastrostomy and antibiotic and sulfa therapy are carried out. The article is well illustrated by x-rays, naked eye views and photomicrographs.

HILL.

MISCELLANEOUS

Treatment With Bacterial Pyromen.

Samter, Max, and Kofoed, Martha A.: *The J. of Allergy* 23:327-334 (July) 1952.

Pyromen, a product of Baxter Laboratories, is a polysaccharide from a pseudomonas species which has been offered for the treatment of allergic diseases. This is because its mechanism of action seems similar to ACTH and adreno-cortico steroids in regard to formed elements of the blood stream. An investigation as to its clinical value and effect on experimental animals was carried out by the authors. In their summary they remark that the effect of Pyromen on sensitization and reinjection with anaphylactic antigens failed to produce a demonstrable decrease in the formation of anti-bodies, or in the results of antigen-antibody reaction.

Clinically, the administration of Pyromen to 38 patients with allergic disease of the anaphylactic type failed to indicate that the compound is superior to a placebo of Pyromen solvent, i.e. 1/6 molar sodium lactate.

VAN ALYEA.

Bronchogenic Carcinoma.

Salvestrini, H., Lucchini, A., and Marsano, A.: *Rev. De Otorinolaryng* 11:1 (April) 1951.

Seventy three cases of bronchogenic carcinoma are reviewed. They are all from a single institution, viz., the department of chest

surgery of the Catholic University Clinic Hospital of Santiago. In sixteen instances the diagnosis was verified. Absolute proof was lacking in fifty seven cases.

The authors estimate that the lesions grow toward one of the bronchi in about eighty five percent of cases. In this group diagnosis is thus facilitated. In peripheral lesions symptoms occur late and are frequently suspected only after metastases are evident. In their experience radiographic evidence was not as satisfactory as cytological examination. Cough is the most frequent symptom. Most frequently it is productive and in about twenty percent hemoptysis was present.

In forty six cases the disease had progressed beyond the possibility of operative relief at the time of diagnosis. Thoracotomy was performed on twenty seven patients.

The authors conclude with the following generalizations: 1. Bronchoscopy is of greatest importance. 2. Cytologic study greatly aids in the diagnosis. Roentgenographic study should be carefully evaluated along with the bronchoscopic findings. 4. Resection alone can offer a cure if done in time. Thoracotomy is ineffective and advocated only as a palliative procedure.

HIGBEE.

Books Received

Progress in Ophthalmology and Otolaryngology: A Quadrennial Review.

Edited by Meyer Wiener, M.D., Alfred E. Maumenee, M.D., Percy Egerton Ireland, M.D., and Joseph Albert Sullivan, M.D., Pp. 668, illustrated, Grune & Stratton, New York, 1952.

Ophthalmic Plastic Surgery.

By Sidney A. Fox, M.S. (Opbth.), M.D., F.A.C.S., Assistant Clinical Professor of Ophthalmology, New York University Post-Graduate Medical School, Pp. 290, illustrated, Grune & Stratton, New York, 1952.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examination:

April 21-25, 1953, in New Orleans, La., at Roosevelt Hotel.

DEAN M. LIERLE, M.D., Secy.
University of Iowa
Iowa City, Iowa

UNIVERSITY OF ILLINOIS

For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

FIFTH INTERNATIONAL CONGRESS OF
OTO-RHINO-LARYNGO-BRONCHO-ESOPHAGOLOGY

The International Committee for the International Congress of Oto-Rhino-Laryngology has invited the Dutch Oto-Rhino-Laryngological Society to organize the Fifth International Congress in the Netherlands.

This Congress will take place on June 8-15, 1953 in Amsterdam.

For further information please address W. H. Struben, J. J. Viottastraat 1, Amsterdam-Z.

NOTICE

Fourth Pan-American Congress of Oto-rhino-laryngology and Broncho-esophagology, Mexico City, January 1954. Dr. Ricardo Tapia Acuna, President, Dr. Chevalier L. Jackson, Executive Secretary, 1901 Walnut St., Philadelphia 3, Pa.

ANNALS

The management of the ANNALS desires to buy, at \$1.50 each, copies of the following numbers which are out of print:

March, 1940

March, 1950

December, 1941

March, 1951

March, 1949

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Derrick Vail, 700 N. Michigan Blvd., Chicago 11, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14, Mass.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Louis H. Clerf, 1530 Locust St., Philadelphia, Pa.
Secretary: Harry P. Schenck, 326 So. 19th St., Philadelphia, Pa.
Meeting: New Orleans, La., April 26 and 27, 1953.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Francis LeJeune, Prytania and Olive Sts., New Orleans, La.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: New Orleans, La., April 28-30, 1953.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOTOLOGY AND RHINOLOGY

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Albert C. Furstenberg, University of Michigan Hospital, Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago, Ill.
Meeting: New Orleans, La., May 1-2, 1952.

THE AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Hugh A. Kuhn, M.D., 112 Rimbach Street, Hammond, Ind.
Sec'y-Treasurer: Joseph W. Hampsey, M.D., 806 May Bldg., Pittsburgh, Penna.
Annual Meeting: October 17, Palmer House, Chicago, Ill.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Secretary-Treasurer: Howard P. House, 1136 W. Sixth St., Los Angeles, Calif.
Meeting: Los Angeles, Calif., 1953.

**HEARING AIDS ACCEPTED BY THE
COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION
OF THE AMERICAN MEDICAL ASSOCIATION**

(As of October 1, 1952)

Audiotone Model 11
Audicon Models 400 and 415
Audi vox Model Super 67
Aurex Models L & M

Beltone Symphonette Model
Beltone Mono-Pac Model M

Cleartone Model 500
Cleartone Model 700
Cleartone Regency Model

Dahlberg Model D-1
Dahlberg Junior Model D-2
Dysonic Model 1

Electroear Model C

Gem Model V-35
Gem Model V-60
Goldentone Models 25, 69 & 97

Maico UE Atomeer
Maico Quiet Ear Models G & H
Maico Model J
Mears (Crystal and Magnetic)
Aurophone Model 200
1947-Mears Aurophone Model 98
Micronic Model 303
Micronic Model "Mercury"
Micronic Star Model
Microtone Audiomatic T-5
Microtone Classic Model T-9
Microtone Model T-10
Microtone Model T612
Microtone Model 45

National Cub Model (C)
National Model D (Duplex)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506

Otarion Model E-4
Otarion Models F-1, F-2 and F-3
Otarion Models G-2 and G-3

Paravox Model D "Top-Twin-Tone"

Paravox Model J (Tiny-Myte)
Paravox Model XTS ('Xtra-Thin)
Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Radioear Permo-Magnetic (Multipower)
Radioear Permo-Magnetic (Uniphone)
Radioear All-Magnetic Model 55
Radioear Model 62 Starlet
Radioear Model 72
Rochester Model R-1
Rochester Model R-2

Silvertone Model J-92
Silvertone Model P-15
Solo-Pak Model 99
Sonotone Model 700
Sonotone Model 900
Sonotone Models 910 and 920
Sonotone Model 925
Sonotone Model 940
Sonotone Model 966
Super-Fonic Hearing Aid

Televox Model E
Telex Model 97
Telex Model 99
Telex Model 200
Telex Model 300-B
Telex Model 400
Telex Model 500
Telex Model 1700
Tonamic Model 50
Tonemaster Model Royal
Tonemaster Cameo Model

Unex Midget Model 95
Unex Midget Model 110
Unex Models 200 and 230

Vacolite Models J and J-2

Western Electric Models 65 and 66

Zenith Model 75
Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)
Aurex Semi-Portable

Precision Table Hearing Aid
Sonotone Professional Table Set Model 50

Index of Authors

- ANDREWS, A. H., 1181.
- BALLENGER, H. C., 542.
- Ballenger, J. J., 542.
- Baltzell, W. H., 677.
- Beaton, A. H., 1206.
- Beck, A. L., 515.
- Benedict, E. B., 1120.
- Blassingame, C. D., 483.
- Boies, L. R., 836.
- Boles, R. G., 1046.
- Boyden, G. L., 558.
- CLERF, L. H., 5.
- Cody, C. C. III, 384.
- Conley, J. J., 62.
- Curry, E. T., 389.
- DAVIDSON, M., 1046.
- Derlacki, E. L., 179.
- Dickson, J. C., 656.
- Dietrich, H. J., 395.
- Dietrich, H. J. Jr., 1134.
- Dix, M. R., 987.
- Dorenbusch, A. A., 83.
- EDWARDS, T. M., 159.
- Emerson, E. B. Jr., 278.
- Engström, H., 1027.
- FENTON, R. A., 470.
- Figi, F. A., 1048.
- Fischer, N. D., 198, 352.
- Fowler, E. J., Jr., 888.
- Freeman, E., 317.
- Furstenberg, A. C., 692.
- GOLDMAN, J. L., 120.
- Goltz, N. F., 441.
- Goodhill, V., 778.
- Goodof, I. I., 474.
- HALLPIKE, C. S., 987.
- Hardy, A., 317.
- Harkins, W. B., 663.
- Hawkins, J. E. Jr., 789.
- Heatly, C. A., 1206.
- Hendrick, J. W., 1094.
- Henry, G. A., 1114.
- Hilding, A. C., 354, 371, 648.
- Hill, F. T., 474, 751.
- Hoffert, W. R., 317.
- Holinger, P. H., 1159.
- Holmes, E. M., 45.
- Hoover, W. B., 1148.
- Hyde, R. W., 937.
- IRWIN, T. M., 408.
- JOHNSON, H. A., 1048.
- Johnston, K. C., 1159.
- Judge, A. F., 625.
- KELEMEN, G., 45, 457.
- Keys, R. J., 317.
- Kimura, R. S., 697.
- Kobrak, H. G., 1053.
- Koons, E. O., 751.
- Kos, C. M., 810.
- LAMBERT, E. H., 850.
- Latella, P. D., 266.
- Lawrence, M., 717, 824.
- Lederer, F. L., 126.
- Lempert, J., 717.
- Lever, W. F., 1120.
- Lindsay, J. R., 697.
- Loch, W. E., 198.
- Loughead, J. R., 154.
- Lurie, M. H., 789.
- MARCUS, M. D., 18.
- Marcus, R. E., 126.
- Makart, C. D., 490.
- Markle, D. M., 888.

- McCart, H., 593.
McCuiag, D. R., 144.
McLemore, C. S., 242.
Mitchell, R. B., 317.
Moersch, H. J., 976.
Morrison, L. F., 567.
Moulonguet, H., 888.

NASH, C. S., 747.
Neff, W. D., 697.

O'KEEFE, J. J., 1071.
Overholt, R. H., 1198.
Owens, H., 435.

PARCHET, V., 1159.
Pollack, R. S., 96.
Priest, R. E., 651, 1039.
Prigal, S. J., 206.
Putney, F. J., 452, 677.

RAMBO, J. H. T., 717.
Reed, G. F., 112.
Reger, S. N., 810.
Richards, L., 1134.
Roberts, W. E., 83.
Rosen, S., 448.

SALINGER, S., 533.
Sataloff, J., 107, 350.
Schall, L. A., 395.
Schuknecht, H. F., 697.
Seltzer, A. P., 276, 1067.
Senturia, B. H., 18, 331.

Shambaugh, G. F. Jr., 873.
Singer, D. E., 317.
Snitman, M. F., 1076.
Snyderman, S. C., 1046.
Somers, K., 636.
Sweet, W. H., 45.

TAMARI, M. J., 1017.
Taylor, H. M., 465.
Titcher, L. L., 400.

VAN ALYEA, O. E., 490.
Vilstrup, G., 189.
Vilstrup, T., 189.

WALKER, J. H., 1198.
Wattleworth, K. L., 656.
Weille, F. L., 90.
Weisman, E. B., 1017.
Weller, W. A., 1080.
Wersäll, J., 1027.
Wever, E. G., 717, 824.
Wiesskopf, A., 681.
Williams, H. L., 497, 850.
Winston, J., 350.
Wishart, D. E. S., 505, 762.
Withers, B. T., 656.
Wolff, D., 717.
Woltman, H. W., 850.

YOUNGER, L. I., 836.

ZERFFI, W. A. C., 642.
Zimmermann, A. A., 1159.

Index of Titles

- ACOUSTIC Trauma in Children. Joseph Sataloff, 107.
- Adult and His Hearing Problem. Francis L. Lederer and Richard E. Marcus, 126.
- Aerosols, Steam Generated, Clinical Application of. Samuel J. Prigal, 206.
- Aerotitis Media. Reed W. Hyde, 937.
- Agenesis of Lung with Tracheal Stenosis. F. Johnson Putney and William H. Baltzell, 677.
- Air Passages, Obstruction of. Wendell A. Weller, 1080.
- Airplane Noise and Auditory Thresholds. Ben H. Senturia, 331.
- Allergy, Aural Manifestations of. Eugene L. Derlacki, 179.
- American Broncho-Esophagological Association, Scientific Papers of, 1114 et seq.
- American Laryngological Association, Scientific Papers of the, 465 et seq.
- American Otological Society, Scientific Papers of, 692, et seq.
- Anesthetic, Local, in Post-Tonsillectomy Pain. Morris Davidson, Robert G. Boles and Sanford C. Snyderman, 1046.
- Anomalies of the Sound Conducting Apparatus and Their Surgical Correction. George E. Shambaugh, Jr., 873.
- Asthmatic, Unsuspected Bronchiectasis in the. Richard H. Overholt and James H. Walker, 1198.
- Auditory Rehabilitation. Frederick T. Hill and Elizabeth O. Koons, 751.
- Aural Approach to the Parotid Gland. Francis L. Weille, 90.
- Aural Manifestations of Allergy. Eugene L. Derlacki, 179.
- Autonomic Dysfunction, Cause of Head and Face Pain. Neil F. Goltz, 441.
- Audiometer Weber Test and Masking. Donald M. Markle, Edmund P. Fowler, Jr. and Henri Moulouguet, 888.
- Audiometric Thresholds in Spring and Fall. E. Thayer Curry, 389.
- BACTERIAL and Cytological Diagnosis of Nasal Disease. Joseph L. Goldman, 120.
- Blind Bouginage for Esophageal Obstruction. Lyman Richards and Herbert J. Dietrich, Jr., 1134.
- Bronchial Movements Observed with Recording Bronchial Caliper. Albert H. Andrews, Jr., 1181.
- Bronchiectasis in the Asthmatic. Richard H. Overholt and James H. Walker, 1198.
- Branchiogenic Carcinoma of the Larynx. Frederick T. Hill and Irving I. Goodof, 474.
- Bronchography in Children. D. E. Staunton Wishart, 505.
- Bronchography in Children. Robert E. Priest, 651.
- Bronchoscopes, Simplified. Ernest B. Emerson, Jr., 278.
- CANCER, Head and Neck, Radical Surgery for. Robert S. Pollack, 96.
- Cancer, Intraoral, Surgery in. Maurice F. Snitman, 1076.
- Cancer of the Larynx, Terminal Phases and Death. Peter D. Latella, 266.
- Carcinoma, Branchiogenic, of the Larynx. Frederick T. Hill and Irving I. Goodof, 474.
- Cardia and Esophagus, Differential Diagnosis. Herman J. Moersch, 976.
- Cartilage Homografts, in Rhinoplasty. Samuel Salinger, 533.
- Chemotherapy in Deep Neck Infections. August L. Beck, 515.
- Chicago Laryngological and Otological Society, 280, 282, 601, 903, 908, 1216, 1220.
- Chisel, Nasal. A. P. Seltzer, 276.
- Cilia, Structure of. H. Engström and J. Wersäll, 1027.

- "Clocking" of the Naris in Demonstrating Rhinoplasty. A. P. Seltzer, 1067.
- Cochlea Function of Under Dramamine. Julius Winston and Joseph Sataloff, 350.
- Cochlea, Sound Conduction in. Ernest Glen Wever and Merle Lawrence, 824.
- Cochlear Aqueduct and Endolymphatic Sac, Obliteration of. J. R. Lindsay, H. F. Schuknecht, W. D. Neff and R. S. Kimura, 697.
- Cortisone in Lye Burns of the Esophagus. Alex Weisskopf, 681.
- Cortisone in Otorhinolaryngology. Henry L. Williams, 497.
- Cysts of the Thyroglossal Duct Complicating Laryngeal Operations. Frederick A. Figi and Hugh A. Johnson, 1048.
- DISPLACEMENT by "Reverse Valsalva." George F. Reed, 112.
- Dramamine, Effect of Upon Cochlear Function. Julius Winston and Joseph Sataloff, 350.
- ENDOLYMPHATIC Sac and Cochlear Aqueduct, Obliteration of. J. R. Lindsay, H. F. Schuknecht, W. D. Neff and R. S. Kimura, 697.
- Esophagitis. Walter B. Hoover, 1148.
- Esophagus and the Cardia, Differential Diagnosis. Herman J. Moersch, 976.
- Esophagus, Lye Burns of. Alex Weisskopf, 681.
- Ethmoid Sinus, Osteoma of. Marvin J. Tamari and Edward B. Weisman, 1017.
- External Ear, Classification of Diseases of. Ben H. Senturia and Morris D. Marcus, 18.
- FACIAL Bone, Fractures of. Carl S. Mc-Lemore, 242.
- Fat in the Tracheobronchial Tree. Alexander H. Beaton and Clyde A. Heatly, 1206.
- Foreign Bodies in Air and Food Passages. Louis H. Clerf, 5.
- Foreign Body, Bronchoscopic Removal of with Triangulation. Alfred A. Dorenbusch and Wendell E. Roberts, 83.
- Fractures of the Facial Bone. Carl S. Mc-Lemore, 242.
- GLOMUS Jugulare Tumor of Middle Ear. Samuel Rosen, 448.
- Grafting of Radical Mastoid Cavities. Ben T. Withers, J. Charles Dickson and Kent L. Wattleworth, 656.
- HEAD Injuries, Tracheotomy in. Howard McCart, 593.
- Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association, 314, 623, 934, 1234.
- Hearing Defects in Children. D. E. S. Wishart, 762.
- Hearing Loss Patterns in Rural School System. E. Thayer Curry, 389.
- Hemangiomas of the Frontal Bone. Edgar M. Holmes, William H. Sweet, and George Keleman, 45.
- Hematoma of the Larynx. F. Johnson Putney, 452.
- Hemifacial Spasm, Synkinesis and Contracture in. Henry L. Williams, Edward H. Lambert and Henry W. Woltman, 850.
- Historical Aspects of Foreign Bodies in the Air and Food Passages. Louis H. Clerf, 5.
- History of Tracheotomy. Robert E. Priest, 1039.
- INFERIOR Alveolus, Tumors, Treatment of. John J. Conley, 62.
- Inferior Cochlear Vein, Experimental Occlusion of. H. B. Perlman, 33.
- Implant, Plastic, for Reconstruction of Nasal Bridge. A. C. Hilding, 648.
- Iodism. Thomas M. Irwin, 408.
- LARYNGEAL Nerve, Superior, Motor Function of. Newton D. Fischer, 352.
- Laryngeal Paralysis, Bilateral, Surgical Treatment of. Thomas M. Edwards, 159.
- Laryngeal Tuberculosis. Ralph A. Fenton, 470.
- Laryngocoele, Surgical Treatment of. John J. O'Keefe, 1071.
- Larynx, Cancer of, Terminal Phases and Death. Peter D. Latella, 266.

- Larynx, Hematoma of. F. Johnson Putney, 452.
- Larynx, Malignant Melanoma of. John R. Loughhead, 154.
- Lingual Thyroid. Herbert J. Dietrich and LeRoy A. Schall, 395.
- Lipoma of the Bronchus. Alexander H. Beaton and Clyde A. Heatly, 1206.
- MALFORMATIONS, Congenital, of Trachea, Bronchi and Lung. Paul H. Holinger, Kenneth C. Johnston, Victor Parчет and Arnold A. Zimmermann, 1159.
- Masking and the Audiometer Weber Test. Donald M. Markle, Edmund P. Fowler, Jr. and Henri Moulouquet, 888.
- Mastoid Cavities, Skin Grafting of. Ben T. Withers, J. Charles Dickson and Kent L. Wattleworth, 656.
- Maxillary Sinus, Rhabdomyosarcoma of. Duncan R. McCuaig, 144.
- Measurements of Recruitment. Scott N. Reger and C. M. Kos, 810.
- Melanoma, Malignant, of the Larynx. John R. Loughhead, 154.
- Meniere's Disease, Anatomical Considerations of. A. C. Furstenberg, 692.
- Meniere's Disease, Pathology and Symptomatology. Julius Lempert, Dorothy Wolff, J. H. T. Rambo, Ernest Glen Weaver and Merle Lawrence, 717.
- Mixed Salivary Gland Tumor. Claude C. Cody, III., 384.
- Motor Function of the Superior Laryngeal Nerve. Newton D. Fischer, 352.
- Mouth, Treatment of Malignancy in. James W. Hendrick, 1094.
- Malignancy in the Mouth. James W. Hendrick, 1094.
- NASAL Disease, Bacterial and Cytological Diagnosis. Joseph L. Goldman, 120.
- Nasopharynx, Radium Treatment of. Walter E. Loch and Newton D. Fischer, 198.
- Neck Infections, Deep, Chemotherapy in. August L. Beck, 515.
- Noise, Airplane, Influence on Auditory Thresholds. Ben H. Senturia, 331.
- Noise in Industry, Medical Legal Implications. C. Stewart Nash, 747.
- Neurilemmoma of Pharynx. Kenneth Somers, 636.
- OBITUARY: Marvin Fisher Jones, M.D., 917.
- Obstruction, Esophageal, Blind Bouginage in. Lyman Richards and Herbert J. Dietrich, Jr., 1134.
- Obstruction of the Air Passages. Wendell A. Weller, 1080.
- Occlusion of the Inferior Cochlear Vein. H. B. Perlman, 33.
- Organ of Corti, Theory of the Stimulation of. A. C. Hilding, 371.
- Osteoma of the Ethmoid Sinus. Marvin J. Tamari and Edward B. Weisman, 1017.
- Otic Labyrinth: Origin and Insertion of the Tectorial Membrane. A. C. Hilding, 354.
- Otitis Externa, Bacteriology and Mycology. David E. Singer, Elizabeth Freeman, Warren R. Hoffert, Reginald J. Keys, Roland B. Mitchell and Albert Hardy, 317.
- Ototoxicity of Streptomycin. J. E. Hawkins, Jr., M. H. Lurie, 789.
- PAIN, Head and Face, Due to Autonomic Dysfunction. Neil F. Goltz, 441.
- Pain, Head and Face, of Sinus Origin. Harold Owens, 435.
- Papillary Sinusitis. A. F. Judge, 625.
- Paralysis, Recurrent Laryngeal Nerve. Lewis F. Morrison, 567.
- Parotid Gland, Aural Approach to. Francis L. Weille, 90.
- Pemphigus, Stenosis of the Esophagus in. Edward B. Benedict and Walter F. Lever, 1120.
- Petrous Bone, Removal for Investigation. George Kelemen, 67.
- Pharynx, Neurilemmoma of. Kenneth Somers, 636.
- Pneumatization, Abnormal of the Temporal Bone. Lawrence R. Boies and L. Ian Younger, 836.

- President, American Laryngological Association, Address of. H. Marshall Taylor, 465.
- Prosthesis, Endotracheal Metallic. William Blake Harkins, 663.
- Prosthesis in Treatment of Deafness. H. G. Kobrak, 1053.
- RADICAL Surgery in Head and Neck Cancer. Robert S. Pollack, 96.
- Radium in Nasopharyngeal Treatment. Walter E. Loch and Newton D. Fischer, 198.
- Recording Bronchial Caliper. Albert H. Andrews, Jr., 1181.
- Recruitment, Measurements and Implications of. Scott N. Reger and C. M. Kos, 810.
- Recurrent Laryngeal Nerve Paralysis. Lewis F. Morrison, 567.
- Rehabilitation, Auditory. Frederick T. Hill and Elizabeth O. Koons, 751.
- Rhabdomyosarcoma of the Maxillary Sinus. Duncan R. McCuaig, 144.
- Rhinolith. O. E. Van Alyea and Carl D. Makart, 490.
- Rupture of the Trachea with Stenosis. G. Arnold Henry, 1114.
- SALIVARY Gland Tumor, Mixed. Claude C. Cody, III, 384.
- Scleroma Cured by Streptomycin. Leon L. Titcher, 400.
- Sinusitis, Chronic Frontal, Surgical Treatment. Guy L. Boyden, 558.
- Sinusitis, Papillary. A. F. Judge, 625.
- Sinusitis, Vacuum Frontal. John J. Ballenger and Howard C. Ballenger, 542.
- Sound Conduction in the Cochlea. Ernest Glen Wever and Merle Lawrence, 824.
- Stenosis of the Esophagus in Pemphigus. Edward B. Benedict and Walter F. Lever, 1120.
- Streptomycin, Ototoxicity of. J. E. Hawkins, Jr., M. H. Lurie, 789.
- Streptomycin, Treatment of Scleroma. Leon L. Titcher, 400.
- Suprahyoid Approach to Base of Tongue. Charles D. Blassingame, 483.
- Synkinesis and Contracture in Hemifacial Spasm. Henry L. Williams, Edward H. Lambert and Henry W. Woltman, 850.
- TECTORIAL Membrane, Origin and Insertion of. A. C. Hilding, 354.
- Temporal Bone, Abnormal Pneumatization of. Lawrence R. Boies and L. Ian Younger, 836.
- Test for Tinnitus Identification. Victor Goodhill, 778.
- Thyroglossal Duct, Cysts of. Frederick A. Figi and Hugh A. Johnson, 1048.
- Thyroid, Lingual. Herbert J. Dietrich and LeRoy A. Schall, 395.
- Tinnitus in Identification Test. Victor Goodhill, 778.
- Trachea, Rupture of, with Stenosis. G. Arnold Henry, 1114.
- Tracheal Stenosis with Agenesis of Lung. F. Johnson Putney and William H. Baltzell, 677.
- Tracheotomy, History of. Robert E. Priest, 1039.
- Tracheotomy in Head Injuries. Howard McCart, 593.
- Triangulation Roentgenoscopy, Bronchoscopic Removal of a Foreign Body with. Alfred A. Dorenbusch and Wendell E. Roberts, 83.
- Tuberculosis of the Larynx. Ralph A. Fenton, 470.
- Tumors of the Inferior Alveolus. John J. Conley, 62.
- UTRICULAR Otolithic Membrane, Movement with Postural Change. Grethe and Thure Vilstrup, 189.
- VACUUM Frontal Sinusitis. John J. Ballenger and Howard C. Ballenger, 542.
- Vestibular System, Pathology, Symptomatology and Diagnosis. M. R. Dix and C. S. Hallpike, 987.
- Voice Production. William A. C. Zerffi, 642.

FO
ANN

UNIVERSITY
OF MICHIGAN

JUL 22 1953

MEDICAL
LIBRARY

THE
ANNALS
OF OTOTOLOGY
RHINOLOGY &
LARYNGOLOGY

VOLUME LXI

DECEMBER, 1952

NUMBER 4

FOUNDED IN 1892 BY JAMES PLEASANT PARKER
ANNALS PUBLISHING CO. ST. LOUIS 1, MISSOURI

THE ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY

Published Quarterly by

THE ANNALS PUBLISHING COMPANY, St. Louis, 1, U. S. A.

Entered at the Postoffice, St. Louis, Mo., as Second-class Matter.

THE ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY is published quarterly by The Annals Publishing Company, P. O. Box 1345, Central Station, St. Louis 1, Missouri. Subscriptions and all communications of a business nature should be sent to this address. Manuscripts for publication should be sent to 1010 Beaumont Building, St. Louis, 8, Missouri.

The subscription price in United States, Spain, Central and South America is \$10.00 per annum payable in advance; \$10.20 in Canada, and \$10.80 in all other countries of the postal union. Single copies may be had at the rate of \$3.00 each. Unless otherwise specified, subscriptions will begin with the current number.

In notifying this office of change of address, both the old and the new address should be given.

EDITORIAL OFFICE	✓	1010 BEAUMONT BUILDING, 8
------------------	---	---------------------------

BUSINESS OFFICE	✓	P. O. BOX 1345, CENTRAL STATION, 1
-----------------	---	------------------------------------

Information for contributors will be found on the inside back cover.
--

THE ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY

Published Quarterly by

THE ANNALS PUBLISHING COMPANY, St. Louis, 1, U. S. A.

Entered at the Postoffice, St. Louis, Mo., as Second-class Matter.

TO CONTRIBUTORS:

Original articles and all other material intended for publication, also exchanges and books for review, should be directed to Dr. Arthur W. Proetz, 1010 Beaumont Bldg., St. Louis, 8, Mo.

THE ANNALS may accept for publication original communications relating to otolaryngology and its borderline subjects, case reports, abstracts, book reviews and such letters and announcements as may be of interest to its subscribers at large. While a reasonable inquiry is made into the standing of authors and the authoritativeness of their statements, the editors and publishers can assume no responsibility for them.

Articles are accepted for publication only with the understanding that they appear in no other journal. This does not apply to their inclusion in the published transactions of the various societies.

Manuscripts should be typewritten, on one side of the paper only. They should be double spaced and widely marginated. If the material was presented before a scientific body, a footnote should indicate its name and the place and date of the presentation. Manuscripts should be revised and corrected for spelling, punctuation and grammar. The telegraphic style, omitting articles and conjunctions, sometimes employed for hospital records, is not acceptable for published articles.

References to other published articles must be complete and the data should be set down in the following, now commonly accepted order: author's surname, initials, title of article, journal, volume, page, month and year.

Illustrations essential to the text will be published without cost to the author, but the editors reserve the right to delete illustrations. These, to be acceptable, must be of first quality. Photographs, wash drawings, and shaded pencil drawings are reproduced by means of half-tone plates. Line drawings to be reproduced as zinc etchings must be in black on white paper. Colored ink, blue quadrille rulings and pencil marks in such drawings (except in rare cases) and photographs of charts or other printed matter are not acceptable. Unless the artist's lettering is of the first quality it is preferable to carry reference lines to the margin of the drawing and to have us set the lettering in type. When it is necessary for the sake of clarity to mount several illustrations together, authors are cautioned to bear in mind the proportions of our pages and to mount them accordingly. Each illustration should have written (not clipped) on its back (1) the author's name, (2) the title of the paper, (3) the number of the illustration, and (4) the legend; (5) the TOP should be clearly indicated. Elaborate tables are likely to be confusing. It is usually preferable to substitute several smaller ones.

Proofs will be sent to authors in ample time for correction. If these are not returned, the articles will be printed as corrected by our readers. These are hand proofs and do not indicate the quality of half-tone plates. Authors should see that plates correspond to their legends and that their tops are uppermost. (This is especially important with photomicrographs.)

Reprints will be supplied at rates quoted when proof is sent. Orders must be signed BY THE AUTHOR.